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MYCOTIC INFECTION OF THE BRONCHO-PULMONARY TRACT*

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ANCON, CANAL ZONE

Within recent years there has been such an increase in the number of articles in the literature dealing with mycotic infections that one is led to believe that there has occurred a renaissance of interest in the subject and that this firstborn child of the bacteriological science, medical mycology, so long subordinated to the study of the fission fungi, schizomycetes, or bacteria, is assuming a place of equal importance in medical thought.

Mycotic infections in mankind affect many structures of the body. Among the most important locations affected are the tissues of the respiratory tract. It is the latter that will be considered in this article.

Mycotic infection of the lungs is more prevalent than is generally suspected, and should always be considered in patients with pulmonary symptoms resembling tuberculosis in which there has been a consistent absence of tubercle bacilli in the sputum.

The diagnosis of primary mycotic infection of the broncho-pulmonary tract is often not made until postmortem observations

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and microscopic examinations of the tissues reveal the true nature of the infection, the presence of fungi in the sputum during life having been considered contaminants of no clinical significance.[28]

Extensive data have accumulated in the course of the last 100 years since the basic work of Berg regarding the etiology of pulmonary mycosis appeared. A series of definite facts are available at present, and numerous old assumptions have been discarded.[24] The original law of Berg, that the infectious agents of mycotic diseases attack stratified epithelium exclusively, was disproved as early as 1854 by Reubold. Since then, numerous reports have appeared in the literature in which it has been shown that the oidium albicans occasionally affect also mucosae with cylindrical epithelium, and may proliferate on cylindrical epithelium, though it has a predilection for the epithelium of the mouth, bronchus, or the esophagus. Occasionally the oidium albicans may affect the internal organs, as shown for the first time in 1868 by Wagner. Infection of the pulmonary tissue by the oidium albicans is unusually rare; it is so rare that some authorities have even spoken of the immunity of the lungs toward this fungus. A case reported by Sattler showed invasion with the oidium albicans to have occurred by way of the esophagus and bronchial path, with the production of a large hemorrhagic infarct in the middle lobe of the lung. A suppurative sequestration of the infarct occurred with almost complete destruction of the visceral pleura overlying the infarct.[24]

For clinical purposes, the fungi invading the broncho-pulmonary system are conveniently classified into (1) yeast-like organisms, cryptococci, oidia, monilia, saccharomyces and endomyces; and (2) filamentous forms, such as the actinomyces group.[28]

Fungi are responsible for the flavor, good and bad, of dairy products and beverages. The bad taste of butter, the foaminess of milk, and the unpleasant flavor of certain cheeses, are a few of the ill effects of contamination with fungi.[29] Grain rust, smut of grain, plant rot, the disease of wheat known as ergot, and others, far too many to enumerate, are due to organisms belonging to the fungus group. They also have a place in industry, in the manufacture of cheese, soy bean sauce, and other products.[10]

Fungi have been found in the sputum of patients in whom the primary infection was in the lungs, in which there was no antecedent involvement of other structures. Yeast-like fungi have been isolated from the sputum in patients suffering from other clinical conditions, such as tuberculosis or carcinoma, in which the fungus

was probably a secondary invader in tissue of lowered resistance or a contaminant. The repeated finding of a fungus in sputum from a patient suffering from either a mild or a severe inflammatory process in the lung is strong presumptive evidence of a fungus infection of that organ. While this finding is not, of itself, sufficient to make a diagnosis of primary infection, the absence of any other definite etiological evidence gives strong support to the probability of such a diagnosis being correct. In some of the patients studied by other observers, the micro-organisms isolated from the broncho-pulmonary lesions have produced typical changes in susceptible laboratory animals.

GENERAL SYMPTOMS OF THE VARIOUS PULMONARY MYCOSES

The mycotic infection in the lung may run a mild course, in which the general condition of the patient remains good, with little fever, a few chest symptoms, the clinical picture resembling a chronic bronchitis. These cases frequently clear up without complications. But there is another more serious type which runs a malignant course. The patient loses weight, and has a cough with hemoptysis. The sputum frequently has a nauseating foul odor which is helpful in making a diagnosis. The physical signs and character of the disease resemble that of tuberculosis. Examination of the chest may show rales and patches of dullness. X-ray plates cannot differentiate between the two diseases because the changes are so similar. A final diagnosis depends upon aspirating affected exudate with the bronchoscope and developing cultures upon specially prepared media.

The average type of fungus infection of the lungs and bronchi resembles tuberculosis and is often mistaken for it. The characteristic finding in most of these infections is the disproportion between the symptoms and the amount of pathological change. The cough is usually only moderately severe, without a large amount of expectoration; a possible fever, and the leucocyte count is seldom more than 10,000. A history of influenzal infection or pneumonia several years before the onset is often encountered suggesting the importance of lowered tissue resistance in its production.[28]

During the past five years there have been 350 cases referred to me for diagnostic bronchoscopy, which have comprised cases of bronchitis, silicosis, pulmonary tuberculosis, and asthma. At the time of the endoscopic examination, routine cultures were taken from each of the large bronchi and examined for fungi. A survey of all cases reported in the hospital since 1919 reveals that there have been numerous clinical diagnoses of bronchiectasis and pulmonary tuber-

culosis, without the presence of tubercle bacilli in the sputum; in a certain number of which, postmortem examination revealed pulmonary mycosis. Roentgenograms of mycotic or pulmonary tuberculosis suspects which did not have tubercle bacilli in the sputum are available on all these cases. The clinical histories of eleven cases are reported in detail as they illustrate the characteristic changes present in such cases reported in this article. There is a difference in the pathological picture presented by the various mycoses in their invasion of the broncho-pulmonary system which will be evident in a detailed analysis of the findings in the several principal types of this group of infections.

Actinomycosis: No group of micro-organisms presents so much difficulty in classification as that now generally designated as actinomycetes. They are mold-like fungi which are normally non-septate and either lack nuclei or possess nuclei too minute to be resolved by the microscope. They reproduce by conidia formed in chains at the tips of aerial hyphae, and also in many cases by a fragmentation of the mycleium into segments analogous to the oidia of higher fungi.

The actinomycetes are an important group of fungi, and at present are classified by many as one of the infections due to higher bacteria, and is included under the streptothrix infections.[333] Such infections are also designated as nocardial. A number of species are pathogenic to man and animals. Lumpy jaw in cattle is annually a serious cause of economic loss. A similar disease in man is relatively frequent and has a high mortality. Madura foot is an important disease in the tropics. In addition to these well known actinomycoses, a large number of other infections due to various species of actinomyces have been reported. Some of these are caused by a closely related group of acidfast forms which produce a disease very similar to tuberculosis. A disease of potatoes, potato scab, and other root crops, is caused by species of actinomyces and is also responsible for great economic losses. The acidfast species of actinomycetes bear a close resemblance to the tubercle bacilli, not only in morphology, but also in cultural characters, pathogenicity, and immunological reactions. Actinomycetes are phylogenetically very close to the mycobacteria and by most bacteriologists are included in the higher group of schizomycetes. The word actinomyces means "ray fungus," and these organisms are frequently referred to in English literature as "ray fungi," and in German literature as "strahlenpilzen." The organisms are present in the lesions they cause and in the pus from them, in the form of spiral or irregularly rounded

grayish or yellowish granulations. When these so-called sulphur granulations, which are a feature of this disease are crushed between slide and cover glass, and examined under high power, one sees an indefinite mass of tangled fibres and debris. On microscopic examination of the involved tissue, pus cells are found at the center of the individual pulmonary lesions, and at the periphery there is a dense network of underlying filaments, which at their extreme margins show the refracting clubs or rays. It is important that one identify both the rays and mycelia before making the diagnosis of actinomycosis.

Lumpy jaw has been well known for half a century. It was one of the diseases first studied by Koch. We are still somewhat uncertain as to the mode of infection and transmission, as to whether the disease may be contracted by man from cattle, for instance, and still a little in doubt as to the nature of the causative organism, whether one or several species of actinomycetes are concerned.

Actinomycetes have been reported as present in the crypts of tonsils, in pus from pyorrhea pockets, and from dental root abscesses. The anaerobic type of Israel is pathogenic to laboratory animals and reproduces the disease in cattle, therefore it appears that the human and bovine cases are caused by this anaerobic type of organism.[23]

Actinomycosis affects many animals, both wild and domesticated. Moody has described lesions, clearly actinomycotic, in the bones of a fossil rhinoceros.[36] The disease is known to occur in deer and moose, sometimes in epidemic form. It has been noted in practically all of the domesticated animals, but mainly in the herbivorous species, and particularly in cattle. The disease in cattle occurs in all parts of the world, but is more frequent in certain areas. Thus in the United States many more cases are observed in slaughter houses of the Middle West than in the Eastern or Southern states.[16]

There is a great deal of evidence to indicate that the infection in cattle is contracted from contaminated hay, straw, or grain. In Europe the disease in cattle appears to be more prevalent in moorlands or in areas that have been flooded. Primary lesions of the lungs and of the intestinal tract also occur in cattle, but are not nearly so frequent as those of the mouth parts. Since infection in cattle is not transmitted from animal to animal, we wonder if the infection in man is due to chewing or inhaling particles of hay, straw, or grain. We often see people going about with hay or straw in between their teeth, chewing nonchalantly a medium probably covered with actinomyces.

Actinomycosis of the pulmonary tissue in mankind is invariably fatal, and although this condition is often confused with tuberculosis, pathologically it is quite different. There is no other disease of the lungs, except perhaps coccidiosis, where there is such tendency on the part of the disease to burrow its way in all directions. Thoracic actinomycosis is caused by organisms aspirated or ingested which lodge in the pulmonary tissue.[33] From a single lesion in the lung, one sees many sinuses radiating in all directions. Involvement of the neighboring organs by direct extension is a striking feature of the disease. Invasion of the pleura and peripleural tissues is almost invariable, and usually leads to the formation of an adhesive type of pleurisy. Erosion of the ribs, perforation of the intercostal spaces in one or more places, perforation of the esophagus, and superficial thoracic abscesses are often found. Invasion of the pericardium and the mediastinum, with erosion of the vertebrae, takes place with regular frequency.

In man, there is evidence that the disease is contracted from infected vegetable matter gaining access to the tissues. Actinomycosis in man is most frequent in adult males, and a very considerable proportion of the cases occur in men engaged in occupations which would expose them to the possibility of inhaling, ingesting, or otherwise having dried vegetable matter introduced into the tissues. Farmers, gardeners, stablemen, tanners, and people engaged in similar occupations comprise more than half the cases. It might be argued that people so engaged are also in contact with cattle and most likely to be infected from that source. The disease is not particularly frequent among slaughter-house employees.[16] In Italy it has been found to occur rather commonly in the men engaged in packing wines in straw. Geographically the disease occurs more frequently in country districts than in cities. It is found in all parts of the world. But in the United States it is clearly most frequent in the cattle districts of the North Central States.

In man the disease has been known to involve all parts of the body, but occurs as a primary lesion most frequently in the neighborhood of the face and neck, next, as primary lesions of the abdominal cavity, and thirdly, as primary lesions in the lung.

Primary pulmonary actinomycosis may develop about particles of inhaled vegetable matter. One case is on record where an awn of barley was found in the center of a primary pulmonary lesion. In some cases, lesions of the lung are secondary to lesions of the mouth parts. Lesions of the lungs vary in character from a subacute bronchopneumonia to a chronic but invariably fatal disease

resembling tuberculosis. In 1920 Henrici and Gardner collected records of 26 cases of actinomycosis in man.[16] In all but three of these the infection was primary in the lungs or peribronchial lymph nodes. In the lungs there is produced a caseous bronchopneumonia with eventually softening and cavity formation, but there is a pronounced tendency for the organisms to become distributed through the blood with the formation of abscesses in other viscera, especially the brain.

Infection with the acidfast type of actinomycetes is to be differentiated from tuberculosis. In pronounced pulmonary cases this is not easily done, and undoubtedly such infections have been mistakenly diagnosed as tuberculosis a number of times. In the sputum the organism readily undergoes fragmentation, and being acidfast the fragments resemble tubercle bacilli very closely. They are more variable in length and sooner or later long branched filaments will be found. Although these forms grow readily on ordinary culture media, they grow more slowly than bacteria, and isolation from sputum by plating is difficult. By guinea pig inoculations pure cultures may be readily obtained. This would seem to be the procedure of choice for establishing the diagnosis. If inoculated intraperitoneally the animal generally dies in less than a week, with very characteristic miliary white nodules over the peritoneal surfaces, especially in the omentum.[36]

In thoracic actinomycosis, early recognition of the lesion is likely to be accidental. Often it is the occurrence of an empyema which is threatening to rupture through the thoracic wall, which gives the first suspicion of the presence of pulmonary actinomycosis. The roentgenologist may occasionally diagnose the lesion when the lung, pleura, and chest wall are involved; that is, when there is an intrapulmonary lesion which simultaneously gives rise to a periostitis, actinomycosis should be suspected. Pulmonary abscess, bronchiectasis with abscess or gangrene of the lung, fungus infection of the lung, broncho-pulmonary spirochetosis, and hydatid cyst, may be confusing to the diagnostician.[33]

Israel[23] has divided the clinical course of the disease into three stages. In the first or broncho-pulmonary stage, the process does not extend beyond the limits of the pulmonary tissue. Incidentally it is in this stage that the condition may be amenable to surgery, for actinomyces are anaerobic, and with the entrance of air which follows the free opening of the many sinus tracts, and the resection of the circumscribed diseased tissue, a complete cure is conceivable. Involvement of the pleura marks the beginning of the second or the pleurothoracic stage, while invasion of the thoracic wall termi-

nates in the third or fistulous stage. This classification presents an instructive picture of the usual progress of the disease. The medical treatment of this condition is one of the gloomy chapters in the history of medicine. In recent years several cases have been reported which have responded to the iodide and copper sulphate therapy. In this disease any form of treatment which promises results is worthy of trial. Because it is not specific, the detection of the presence of actinomycosis by cutaneous reactions similar in character to the Mantaux test for tuberculosis is not feasible.

The literature on broncho-pulmonary diseases reflects a pessimistic outlook in thoracic actinomycosis. The problem with such infections is their early recognition and prompt application of surgical measures, which should comprise evacuation of the exudate and dead tissue, and obliteration of tissue of all the pockets (since it is an anaerobic organism) whenever it is possible to do so. Thus the treatment of actinomycosis has three components: an especially careful sanitation of all infected foci; extraction of the teeth revealed as diseased in the maxillary and mandibular types, as their conservation in the presence of actinomycosis is more than doubtful; and in addition, constitutional treatment of concomitant general diseases (such as tuberculosis and syphilis) with which it may be associated. Evacuation of exudate or dead tissue, and effective irradiation should be employed since lesions are classed with the infective granulomas which are generally regarded as highly radiosensitive. The iodides are of great value internally.[31]

Stewart relates that since 1905, roentgen irradiation has been the choice treatment of actinomycosis.[26] Simple fractional irradiation with relatively high doses (about 4 x 300 r/I, 0.5 Cu.) have given best results. In superficial localization of the process the prognosis is uniformly favorable, especially so in actinomycosis of the head, neck and chest. Between 1922 and 1933, he treated eight cases involving the lungs, the liver, the intestines, or the abdominal walls. One series of treatment was necessary. The total dose varied between 1200-1500 r/I, in most of the cases the treatment was not followed by a dermatitis. Of the other advantages of the protracted fractional irradiation the following was mentioned: the acceleration of the duration of treatment, the elimination of dangerous reactions, and finally the possible elimination of a second series of treatment. Details of the technic vary with each case and will attempt to eliminate the poor results obtained in deep actinomycotic affections. On successive days, once daily is used 240 r/I, 1.3 mm. cu., 60 cm. distance HWS 1.3-1.5 mm. cu., tension at the tube 170 KV, R—

introduction 2.5-3.5 r/I per minute. Roentgen value 4-5 r/I per minute. These doses are repeated 5-8 times. The patient is given at the same time potassium iodide by mouth.

Fried, commenting on the effectiveness of irradiation combined either with surgery or the use of iodides in the treatment of actinomycosis, believes it to be superior to any other.[7] He contends that the conditions are properly classed with the infective granulomas which are generally regarded as highly radiosensitive. The efficiency of irradiation in actinomycosis is due to an indirect action of the rays which modifies the tissues so as to render them poor media for the growth of the organism. Roentgen irradiation was administered to all lesions of the head and neck, given at the site of the disease once or twice weekly, over a period of 69 days. Other factors were 140 kv., 5 ma., and filtration with 4 mm. of aluminum. In the course of the treatment it was noted that although the pathological structure of the palpable borders of the lesion disappeared within ten days, new lesions developed in contiguous areas that had been shielded. Various irradiation technics used by others show ray ranges from 95 to 200 kv., the filtration from 4 mm. of aluminum to 1.3 mm. of copper, and the intensity from 200 to 800 r. Others employ the protracted fractionated technic.[20]

The rationale of surgical treatment lies in the fact that the infection is essentially an anaerobic one requiring removal of dead tissue which is poorly oxygenated and in consequence an excellent culture medium. In extensive cases energetic surgical excision of devitalized tissue is indicated.

Wangensteen's experience with the treatment of thoracic actinomycosis affords no occasion for optimism; yet, if the disease can be diagnosed early and prompt evacuation of the dead tissue effected, means of extending the disease is done away with. In those instances in which the diseased process remains well localized, and particularly if dissection towards the surface occurs, prompt and adequate treatment such as curetting of abscesses should be done.[33]

Medicine: Zinc peroxide, for anaerobic infection, should be useful.[33] The iodides are specific on mycosis.[5] Potassium iodide may be given intravenously to good advantage. In children the syrup of ferrous iodide seems to be equally effective. Trauner suggests injections of a lymph gland extract.[31] This extract is a limpid aqueous solution made from the cervical lymph nodes of cattle. It is injected subcutaneously into the forearm. The dose is 1 cc. The injection is made at intervals of two or three days. The use of this extract is recommended in addition to surgery and other measures by Trauner.

Monilial infection of the broncho-pulmonary system was first described by Castellani in Ceylon in 1905, which occurred in tea sorters. Since then pulmonary moniliasis has been observed, studied, and recorded, by a number of investigators in different parts of the world, especially in the tropical countries.[6] Subsequent studies in Europe and especially in the United States showed that especially in this country bronchomoniliasis of Castellani is much more diffused than has been commonly admitted (Stovall and Greeley, Stokes, Kiser, and Smith).

Pulmonary mycosis caused by fungi of the genus monilia is rarely observed in Italy. The possibility of bronchopneumonial syndromes caused by these agents has been scouted by Castellani and confirmed by others. The importance of monilia has been denied by some authors considering the marked difference of the genus and frequent association with other pathologic processes and also on account of the inconstancy of its morphologic and sometimes also biochemical properties.[18]

Two distinct forms of the infection may be distinguished, i. e., the mild and the severe forms. The clinical symptoms of the first are those of a common bronchial catarrh, while those of the severe form are similar to those in pulmonary tuberculosis. The diagnosis of bronchomoniliasis is based on the findings in the expectoration, absence of the Koch bacillus, absence of pulmonary syphilis, and the pathogenicity of the strain.[10]

The bronchopneumomoniliasis of Castellani is now recognized as a nosologic entity.[6] It has been defined and repeatedly confirmed not only in the tropical countries but also in temperate climes. Cases have been reported from France, England and Greece. Eight or nine cases have been reported in Italy. The clinical symptomalogy is generally identical with that of chronic common bronchitis, with periods of recrudescence, and participation of the pleura and diffusion of the process to the pulmonary parenchyma in some cases. These latter cases are considered as grave in nature, and have frequently been erroneously classified with pulmonary tuberculosis. The pathologic contributions on this subject are few in number and mostly refer to experimental investigation.[18]

Bronchomoniliasis is no longer considered a rare condition, although the diagnosis must be made with extreme reserve, since the organism is frequently demonstrated in the secretions of the upper air passages of a normal individual, and particularly in the sputum of patients suffering from chronic pulmonary disease, notably tuberculosis.[15]

Monilia may be regarded physiologically as true yeast, since they too ferment sugars with the production of gas. Morphologically, they differ from true yeast (saccharomyces) in that they have a negative staining polar body and a mycelium consisting of a collection of fine filaments or threads termed hyphae and reproduce by free-born spores of conidia. The pathogenicity of the monilia depends greatly on the resistance of the host.[5] In man, these organisms may normally occur in the digestive tract without producing demonstrable lesions. Most physicians know of "Thrush" as it affects children and cachetic adults.[1] It is a monilial infection of the oropharynx, and it has been frequently observed that there is a preceding history of respiratory infections prior to the onset of the moniliasis.[11] In the opinion of many observers there is a primary irritating factor, bacterial, toxic, or mechanical, which brings about changes in the bronchi which permit the fungi to gain foothold. However, the lesion having been once established, the clinical picture becomes that of a primary broncho-moniliasis.[17]

Inhalation of contaminated dust or air probably plays the most important role in the transmission of the fungus deep into the respiratory tract, as the monilia are resistant to drying, and may live indefinitely in dust and dry environment.[15]

Monilia may exist as saprophytes in the upper air passages. A yeast-like fungus is frequently observed in the secretions of the nose and throat and the accessory sinuses.[12] Under favorable conditions, the organism invades the lungs. Likewise, monilial lesions of the skin and mucous membrane elsewhere in the body may be a source of infection for secondary foci in the air passages. The transmission from person to person is also a possibility. More than one member of the same family has been known to be afflicted with this condition.

Since fungus requires an acid medium for its growth, there must necessarily be some chemical change in the otherwise alkaline secretion of the bronchus to favor the continued growth of the organism. This suggests the existence of a pre-existing, primary lesion which must alter the chemical reaction of the tissue before the invasion of monilia can take place.[15] A person's dietary habits have some influence in the reaction of the bronchial secretions, and the body chemistry of the patient should be considered in the treatment.

Early primary lesion of bronchomoniliasis has not been described, and probably cannot be distinguished from those caused by any other form of inflammation. It is possible that a primary bac-

terial or allergic inflammation of the bronchi or the pulmonary parenchyma may first prepare the "soil" for the invasion and growth of monilia, which, once firmly planted in the tissue, continues to live a tenacious, semi-saprophytic existence, and to stimulate mildly chronic irritations with a resulting low grade inflammation. This conception is supported by the finding of monilia in the walls of the bronchi and in the regional lymph nodes at necropsy in a case of chronic bronchial asthma of several months duration. Such findings may be considered the pathological basis of bronchomoniliasis in the early stage.

The continued low grade inflammation of the bronchial wall adds susceptibility to a recurrent acute infection probably including small localized areas of pneumonia which undoubtedly favor the more rapid growth of the fungus. This results in the formation of small abscesses of pseudotubercles in the involved areas. These areas of tumefaction which constitute the so-called mycotic tumors probably undergo resolution in the majority of cases, leading to complete recovery. Others may break down, to form true abscesses which may coalesce to form larger ones. Such abscesses may be the basis of the advanced form of bronchomoniliasis, or may eventually heal and be replaced by fibrous tissue. Usually a proliferative pneumonitis results in which the major pathologic lesions include the scattered areas of fibrosis, bronchiectasis and abscess formation with intervening and adjacent zones of atelectasis and emphysema. In other instances, a chronic pneumonia of unknown origin may become infected with pathogenic monilia which by adding insult to injury completes a clinicopathologic picture of bronchomoniliasis.[15] Overwhelming infection with monilia in chronic specific lesions such as advanced pulmonary tuberculosis, may actually bring about complete annihilation of the primary etiologic agent, thereby transforming it to true bronchomoniliasis—a speculation which may well be within the realm of possibility.

Microscopically, as in gross appearance, there are no lesions specific for this condition. More conspicuous are the non-specific granulomatous lesions in which fibroblastic proliferation and cellular exudation are found, the infiltration being usually entirely of plasma cells, less frequently of polymorphonuclears or of mixed cellular elements. Other changes include the peribronchial and perivascular round cell infiltration, proliferative endarteritis and the proliferative reaction of the alveolar epithelium, all denoting a long continued low grade inflammation. Distinct abscesses of bronchiectatic cavities lined with a heavy zone of infiltrates are frequently encountered.

There are also areas predominantly productive in type with spreading interstitial fibrosis which obliterates alveoli and causes atelectasis and emphysema of the adjacent parenchyma. The macrophages are conspicuous in the neighboring less active areas, while foreign body giant cells may be occasionally observed.[15]

The presence of monilia—the nests of yeast-like bodies and mycelia cannot be demonstrated by the routine hematoxylin and eosin stain, which accounts for the universal failure to make the pathologic diagnosis of this condition where such stains are used. The Gram-Weigert stain[15] brings out the fungus very distinctly, and it is emphatically recommended that it be employed in every case presenting obscure chronic suppurative lesion of the lungs, as many diagnostic oversights will be avoided. The yeast-like bodies are scattered through the lesions, particularly in the walls of the abscess and perivascular spaces. They are doubtless taken up by the lymphatics as a pseudo-foreign body. Mycelia are encountered less frequently. The regional lymph nodes are invariably invaded with the fungi.

With the findings of histopathogenic characteristics already enumerated, and in the absence of other specific etiologic agents in the lesion, and in the light of the positive clinical and laboratory findings, a pathologic diagnosis of bronchomoniliasis may be reasonably made.

The sequence of events leading up to the development of this condition may be described as follows: The fungus gains its entrance into the lower air passages and begins to multiply in the inflammatory exudates caused by some pre-existing disease, such as bronchitis, bronchiectasis or pneumonia. The wall of the bronchus may be invaded by the organisms or they may reach the alveoli and involve pneumonic areas, preventing their resolution, and, by continuing to multiply, may produce a low grade suppurative type of inflammation. This sequence of pathological events may continue indefinitely, perhaps for many years, in a vicious circle, and periodic attacks of pneumonia with areas or subsequent fibrosis and suppuration occur.[15] It would appear that a pre-existing, primary lesion in the lung is essential to initiate the infection by the fungus. Whether specific organisms, such as the tubercle bacillus, may not be exterminated by the overwhelming presence of monilia and its metabolic products is an interesting speculation.

The monilia are of comparatively low pathogenicity to man, but often lead a tenacious, saprophytic existence in him.[6] A primary inflammatory lesion in the lung, with its attendant biochemical

changes in the tissues affords a favorable medium for bacterial growth, and when this is coupled with an inherent or acquired susceptibility of the host, the fungus may become pathogenic in the lung of the affected individual and produce a chronic suppurative inflammation designated clinically as bronchomoniliasis.[15]

The pulmonary form of moniliasis is caused by a yeast-like fungus which is classified as monilia albicans. The organism is oval in shape and measures from three to ten microns in diameter; reproduces by budding; and produces septate hyphae which intertwine to form a mycelium. According to Penta, this organism is often found in association with other bacteria in common respiratory infections, and may, if the resistance of the pulmonary tissues becomes markedly lowered, completely overgrow the primary invaders.[23]

Craven is of the opinion that chronic bronchitis and bronchiectasis caused by so-called non-specific organisms occur frequently in medical practice, alone or together, with various degrees of parenchymatous involvement.[2] Most of the cases are of long duration, and are often associated with the distressing symptoms of nonseasonal asthma. The writer is of the opinion that at least some of the cases of this nature are allergic and due chiefly to the absorption of allergens formed during fungus growth with the production of local areas of inflammation and edema.

In an endeavor to fix a more exact etiology for these illnesses the freshly expectorated sputum of each case was studied by Craven as follows:

1. (a) The gross characteristics of the specimen were observed; (b) darkfield examination and gentian violet stains of fresh smears for fuso-spirochetal organisms were made; (c) examination of fresh smears in 10 per cent sodium hydroxide for spores and mycelia is next in order; (d) examination for tubercle bacilli and other organisms by the Ziehl-Neelson method; and (e) Gram stain.

2. (a) Suspected particles of fresh sputa are next streaked on blood-agar slants and incubated at 38 degrees C.; (b) and for fungus culture they are streaked on Saboraud's medium and incubated at 37 degrees C. and 25 degrees C. Cultures for fungi should be observed for two weeks, as some of the members of this group are slow in growth, and Gill believes many diagnostic oversights are traceable to failure to observe this simple rule.[10]

Craven reports seven cases of chronic nontuberculous pulmonary disease in which the aspergillus fumigatus and monilia albicans were obtained from the sputa. In four cases, nonseasonal asthma

was a dominant symptom. Three cases of infection with monilia albicans occurred in one family demonstrating the importance of intimate contact. Craven found erosio-interdigitalis associated with two cases of pulmonary moniliasis in his series. In a direct smear of pus aspirated from one of the nodules on the hand, numerous spores were seen, which on cultivation proved to be monilia albicans. In this case there was no discoverable pulmonary disease.[2]

Bronchomoniliasis may be recognized clinically in one of three forms, the mild, the intermediate, and the severe. Actually, however, all forms of respiratory disorders are seen, from those closely simulating advanced pulmonary tuberculosis and acute lobar pneumonia, to the more mild and chronic form of tracheobronchitis, bronchitis, and asthma. In the milder types toxic manifestions are lacking. In the mild form the patient has a slight cough, without blood in the sputum, without temperature elevation or the appreciable physical signs of pulmonary disease. The sputum is scanty and mucoid in character; the cough may continue for weeks or months. The usual diagnosis of chronic bronchitis is made after the routine elimination of early pulmonary tuberculosis. Frequently recurrence of the symptoms is a characteristic clinical feature.

In the intermediate form, the clinical symptoms and physical signs are more exaggerated. There is a persistent low-grade fever; the cough is more troublesome; the sputum is mucopurulent and tenacious and may be abundant. There is frequent recurrence of symptoms. The diagnosis of chronic bronchitis, bronchiectasis, or bronchial asthma, is usually made, although the possibility of pulmonary tuberculosis is never altogether excluded.

In the severe form two clinical types may be recognized. In the first type, similarity to pulmonary tuberculosis is frequently evident. The patient suffering from the mild or intermediate form of this condition may suddenly develop an acute pneumonia involving a wide area of the lung. This may be a typical form of lobar or bronchopneumonia, or as a diffuse inflammation of the lung, in which pyogenic micro-organisms usually play an associate role, while, at the same time the fungus finds a fertile soil for further multiplication.[15] The patient is acutely ill, with elevation of temperature, and all evidences of acute pulmonary infection. This lasts for a week or more and may subside completely, or may be followed by an empyema or other complications of acute pneumonia.

The second type of the severe form may result from a complication of the preceding type, or may represent a progressive low grade infection of long standing, in which no etiologic agents can

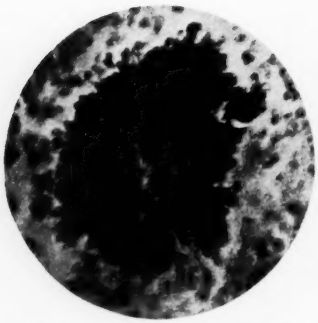

Fig. 1. Ray fungus in actinomycotic abscess of testicle. Fatal case of generalized actinomycosis. Mallory's stain. (X 1200—N.W.)

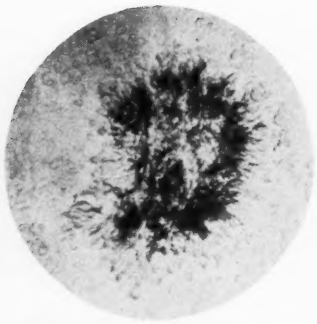

Fig. 2. Ray fungus in actinomycotic abscess of testicle. Fatal case of generalized actinomycosis. Mallory's stain. (X 1200—N.W.)

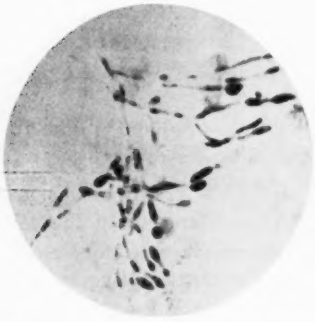

Fig. 3. Monilia albicans in smear from kidney of rabbit dying 48 hours after intravenous inoculation. Gram stain. (X 1000—L.L.H.)

Fig. 4. Monilia albicans from sputum-culture on Sabouraud's medium. Fresh preparation. (X 1500—A.G.R.)

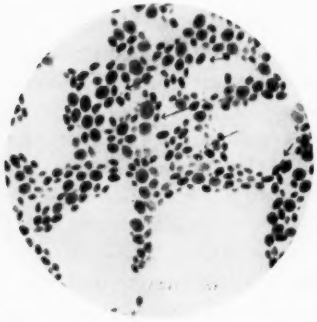

Fig. 5. Photomicrograph of torula in pure culture. The cells in this culture appear principally as the oval form. Several budding forms may be seen. (Reprinted by permission of W. D. Gill.)

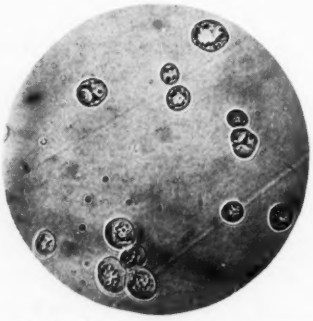

Fig. 6. Saccharomyces from sputum after three weeks' cultivation on McKelvey's carrot infusion—calcium sulphate medium. Ascospores at one and six o'clock. (X 900—A.G.R.)

be demonstrated save the pathogenic monilia which offers the utmost difficulty in differential diagnosis. A diagnosis of chronic advanced pulmonary tuberculosis is often favored in spite of the repeated absence of tubercle bacilli in the sputum. It runs a chronic course, with periods of exacerbation. The patient may run a hectic temperature and complain of night sweats; there is gradual emaciation and loss of weight and strength, and the patient suffers from attacks of dyspnea and severe paroxysmal cough which is worse during the night. The sputum is usually copious in amount, mucopurulent, tenacious, glairy, and often hemorrhagic; its odor may be yeast-like or sweetish.[15] Occasionally, the secondary or co-existing bacterial invaders may greatly alter the character of the sputum. Physical examination elicits signs of patchy consolidation and fibrosis and of bronchiectasis or small cavities, usually limited to the lower lung fields. In the final stages of the disease a gradual failure of the right side of the heart may develop, which is directly responsible for the death of the patient.

The roentgenogram of the lungs merely may show the usual changes of chronic bronchitis or bronchiectasis. In the severe form, there may be widespread shadow, indicative of an acute diffuse pneumonia. More frequently, soft, irregular, mottled or feathery shadows with peribronchial thickening and infiltration and intervening areas of emphysema throughout a greater portion of the lungs are described. Sometimes cavities are present, while the pleura may be thickened in chronic mycotic pneumonia. The mere demonstration of the yeast-like organism of the genus monilia in the sputum does not constitute the diagnosis of bronchomoniliasis.[5] The sputum must be obtained directly from the lungs by bronchoscopy, after every precaution has been taken to prevent contamination from other sources. It is, as a rule, quite tenacious and mucopurulent, and may be blood-tinged or frankly hemorrhagic during the acute stage. It often simulates a typical "asthmatic" sputum in consistence and general appearance. On close inspection, small whitish granules may be observed which represent minute bacilli unless tuberculosis exists as a concomitant infection. There may be a predominance of eosinophiles in the sputum. On stained smears or wet preparations, the yeast-like bodies, budding forms and sometimes branching filaments of the fungus may be demonstrated with comparative ease, especially during the active stage of the disease.[15]

For exact classification the isolated organism should be subjected to the known methods of identification and its pathogenicity determined through animal inoculations. It is generally agreed among medical mycologists that M. Albicans is the only species of the Genus

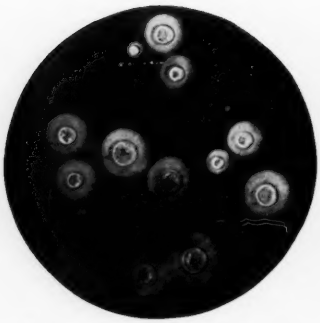

Fig. 7. T. histolytica. Fresh preparation from lung of mouse 16 days after intraperitoneal inoculation. (X 900—J.R.)

Fig 8. C. immitis in lung. Fatal case of coccidioidal granuloma. Hematoxylin and eosin. (X 2500.)

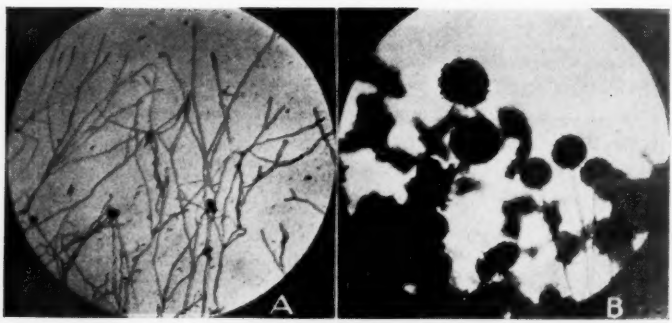

Fig. 9. A. Photomicrograph of penicillium. B. Photomicrograph of aspergillus. (Reprinted by permission of W. D. Gill.)

Monilia which is pathogenic to man and should be regarded as the one responsible for the development of bronchomoniliasis. It is, therefore, imperative that the identity of the isolated fungus be established before the significance of the findings is made known.[15]

Treatment for most of the mycotic diseases consists in giving large doses of the iodides,[22] but some of the cases are resistant and fail to improve under any kind of medication and run a fatal course. Potassium iodide in large doses combined with sodium iodide intravenously seems to be effective.[10] As high as 120 grains of potassium iodide orally in conjunction with 15 grains of sodium iodide has been given daily for a period of two weeks without harmful effect in a patient with a localized torulosis. In children the syrup of ferrous iodide seems to be equally effective. The lesions should be treated with x-ray in conjunction with other therapeutic measures, x-ray to be repeated in five to ten days. Kehoe states that colloidal copper has been used in torulosis because of the similarity of this disease to coccidioidal granuloma in which this drug has acted favorably.[17] Gill, also, advises the use of torula vaccine from which he has observed beneficial results, though he recommends it only on theoretical grounds.[10]

Coccidioidomycosis: The coccidioides, as a pathogenic agent, play a role chiefly in dermatology; of the internal organs the lungs are most frequently involved.[32] Most authors are of the opinion that the pathology of pulmonary coccidiosis is a field regarding which there is still very little known.[9] In the limited literature, clinical symptoms which are similar to those of tuberculosis are described. The true nature of the disease is apparent if a careful analysis is made of the history, with an absence of tubercle bacilli in the sputum, and of the clinical picture of an essentially chronic pulmonary lesion, then the roentgenological examination makes the diagnosis more probable. In such instances one should confirm his suspicion by a search for the coccidioides in the sputum. Skin tests with coccidoidin are of great diagnostic value when properly evaluated.

The fungus coccidioides immitis is round or oval double contoured body, having an appearance somewhat similar to the blastomycetes. It differs, however, in that it contains endospores and reproduces by endosporulation instead of budding. The yeast-like fungus appears in the lesions as an oval or spherical body having a diameter of ten to twenty microns consisting of finely granular protoplasm. It is enclosed in a double contoured, highly refractive hyaline capsule. On examination of the cultures, spherical or oval budding cells, fruit bearing aerial hyphae and mycelium may be

seen. It is more than possible that many cases go unrecognized, since the positive diagnosis of this disease is far from an easy task, requiring time, patience, and considerable special technique for its recognition.

Geographically, the disease has been recognized in South America and Italy, and in about a dozen states of our country.[33] In the San Joaquin Valley, California, coccidioidal granuloma has been found to occur with considerable frequency. Cattle and sheep as well as human beings acquire the disease. There is no indication of transmission of the disease from man to man, animal to man, or vice versa. Persons of all ages and of either sex may be affected. It is known as "Desert Fever" or "Valley Fever."[37] It occurs in highest incidence in regions where there is prolonged hot, dry weather, favorable to the dissemination of the spores.[33] K. F. Meyer states that in Brazil the infection is not aerogenic. In a tropical climate dust infections are infrequent and the South American type is a local process with primary distribution in the buccal cavity—tonsillar invasion and infection of the tongue and intestines. He calls attention to the allergic tests, like those made with coccidiodin, which should be made from several strains of coccidioides to be polyvalent in character. Inhalation is the most likely avenue of infection by the chlamydospores in the vegetative phase of the fungus, though infection might occur through abrasions on the skin. The organism may lodge in the lymph nodes of the hilus region of the lungs, and necroscopy may show a focus of infection obviously older than the other pulmonary lesions, which indicates that they may probably be the primary focus.

Many cases are probably never diagnosed. The disease is thought to begin in the lungs, and though the exact incidence of the infection and the pathological termination of many of the cases is therefore unknown, ordinarily the primary disease is found in the base of the lung, and causes a picture resembling bronchopneumonia, which as it advances, changes to the picture of miliary tuberculosis. In either case the diagnosis can be made only by demonstrating the causative fungus in the discharges from the lung in conjunction with a positive skin test with coccidioidin. The cases resembling bronchopneumonia may clear up in such a manner as to defy differentiation, as the fungus is not universally demonstrable. It is probable that the organism may remain encysted in the lung tissue or adjacent lymph nodes, as foci of coccidioidal infection have been found in supposedly tuberculosis lesions. It is believed by some authorities that there is a tendency to recover from this infection,

which is not invariably, as many believe, always followed by dissemination and speedy death, though this is the general opinion concerning visceral coccidiosis.[33]

From a study of the roentgenograms of many of these cases, Evans and Ball make the following comment: "A diffuse shadow, at times simulating pneumonia, occurs in many cases, and involves the right lower lobe twice as frequently as any other part of the lung field, though it may occur in any portion, and twice as frequently in both hilar regions as in one. In many cases the lesions resemble those of a disseminated miliary tuberculosis."[33]

Infection with coccidioides fungus may be manifested in two ways: by a primary acute infection of the respiratory tract, often accompanied by erythema nodosum, from which the great majority of patients recover without complications; and by a later more or less chronic granulomatous disease, known as coccidioidal granuloma, which is usually fatal with a mortality of 50 per cent, and all attempts at treatment have proved unsatisfactory. The development of granulomas is undoubtedly secondary and is caused by migration of the infectious agent to different parts of the body from the primary foci in the lungs or peribronchial lymph glands, presumably by the blood stream.

The onset is often thought to be a "bad cold" or "flu" with initial symptoms of fatigue, malaise, headache, sometimes pleurisy, or early sore throat, and at times a nonphlyctenular conjunctivitis. Fever may be 100 to 101 degrees F., perhaps 104 to 105 degrees F., and there occurs a rapid loss of weight. Cough comes relatively early and the sputum is usually mucopurulent in character, sometimes streaked with blood, and of variable amounts. The early physical examination of the chest is usually negative or misleading; more often there are symptoms of severe bronchitis or bronchopneumonia. The patient often improves rapidly after four or five days unless there may be an outcropping of lesions of erythema nodosum in about eight days, with associated joint pains. The lesions of erythema nodosum may appear on the thighs, buttocks, arm, upper part of chest and scalp, but the usual site is over the tibial region. Their color may change from fiery red to purple and then fade. The x-ray of the chest in the acute attack shows signs of enlargement of the hilar shadows and shadows of parenchymal involvement. A roentgen diagnosis of tuberculosis is often made on the first examination. The parenchymal shadows usually disappear after six weeks to three months. There is usually clinical recovery after the nodose erythema disappears. The leucocyte count is 8,000 to 15,000 with

eosinophiles 4 to 8 per cent, especially if erythema nodosum is associated. Sphericules of the fungus coccidioides are usually demonstrable in the sputum, and a positive Sabouraud's reaction may be obtained. The urine does not show remarkable changes. In sputum, acidfast bacilli and coccidioides do not occur simultaneously, although patients have been known to have both coccidiodes infection and tuberculosis and continue to exhibit this phenomena. Frequently, all that is found is the spherules of the coccidiodes type with culture of fungus coccidioides and coccidioidal granuloma in injected guinea pigs. It is not yet known how early in the disease the coccidiodes fungus may be found in the sputum or how long it may persist.[37]

The use of the coccidioidin cutaneous test in case of "Valley Fever" has been most helpful in correlating the diagnosis with coccidioides infection. It is a specific skin antigen and extremely active preparation. The reaction is said to be more violent in valley fever than in cases of coccidioidal granuloma. Dickson found that 0.1 cc. of a 1 to 1,000 dilution of coccidioidin is adequate and produces well marked reactions, sometimes with vesicle formation in cases of primary infection.[37] At least 0.1 cc. of a 1 to 10 dilution of this coccidioidin is necessary for coccidioidal granuloma. Dickson no longer uses the control since very allergic types of individuals have failed to react to the control. In cases of primary coccidioides infection without erythema nodosum in which fungus coccidiodes is found in the sputum the diagnosis may be made of bronchopneumonia or influenza.

Dickson advises caution in drawing conclusions in the use of coccidioidin diagnostically.[37] It is necessary to know if the patient previously lived in the San Joaquin Valley or some other region where the infection is common, as sensitivity, and therefore positive reactions persists for long periods after it is once acquired. He also states that erythema nodosum is not exclusively due to coccidioides infection. He has found several people in the San Joaquin Valley with erythema nodosum who were negative for coccidioides infection and proved positive for tuberculosis, as well as at least one from whom the coccidioides spherules and acid-fast bacilli were both recovered from the stomach washings and both coccidioidin tests and tuberculin tests were positive.

Cutaneous manifestations, which are a prominent feature of this disease, may precede the onset of pulmonary symptoms, and in their absence the condition is often confused with tuberculosis. The

absence of tubercle bacilli and the repeated findings of yeast-like cells in the sputum, should make one suspicious of coccidiosis. In general, it may be said that in any case with pulmonary symptoms, expectorating two ounces or more of mucopurulent sputum in 24 hours, tuberculosis can be excluded if repeated examinations are negative for tubercle bacilli.

Blastomycosis (blastomycosis is a generic term and is subdivided into (a) monilia; (b) oidiomycosis; (c) coccidioses; (d) torula; (e) saccharomicosis) and glanders must be also excluded, the former by the presence of budding forms in tissues in contradistinction to the endosporulating fungus coccidioides, and the latter by finding B. Mallei in the secretions. Neither fever, blood count, nor blood cultures are of any value in diagnosis, and there is no record of the recovery of coccidia in the stools or urine. To rule out error in diagnosis, it is best to inject some of the material aspirated from unbroken abscesses into the peritoneal cavity of the male guinea pig, and in about four weeks look for the fungus in pus aspirated from the testicle.[33] It is extremely dangerous to the laboratory worker and bronchoscopist, since Dickson proved that inhalation of culture media can cause infection in the worker.[37]

As infection with the pathogenic yeasts ordinarily referred to as blastomycosis, cutaneous lesions may precede the onset of pulmonary symptoms. However, it is more common in coccidioidal granuloma to have a history of respiratory infection several weeks before the appearance of the external lesion. Trauma seems to be the predisposing factor, and lesions often develop at the site of the traumatized area. Involvement of the epiphyseal ends of the long bones is very common in this condition, and cases develop large fluctuating subcutaneous abscesses from which pus can be aspirated with ease. In the pulmonary form of this disease, the clinical and pathological pictures are difficult to distinguish from those of tuberculosis.

As reported by Morrow and Miller,[36] the treatment of coccidioidal infection has been disappointing, the variable course of the disease must be considered when evaluating the results of any form of treatment. Prolonged observation should temper any enthusiasm for any apparently successful form of therapy. The patient should be treated like a case of active tuberculosis, with emphasis on rest, nutrition and hygiene.

The value of treatment in selected cases:

Therapeutic measures: (1) Surgery which should comprise amputation of local lesions, destruction of local lesions with actual

cautery; drainage of abscesses. (2) Roentgen therapy: this is often of value when applied to local lesions. (3) Drug therapy: no drug has been of constant value. Iodides (potassium iodide 5-6 gms. daily), thymol, tarter emetic, creosote, antimony and potassium tartrate intravenously 1 per cent solution, colloidal copper, salvarsan, and other arsenical preparations, have apparently been of value in some cases. (4) Specific therapy: coccidioidin made from coccidioides has given diagnostic results. (5) Nonspecific therapy: typhoid vaccines, etc., and artificial fever, have been employed with conflicting reports.

Sporotrichosis:[23] The pulmonary form of sporotrichosis is occasionally seen in this country, though by far the greatest number of reported cases have come from Europe. Fifteen cases have been collected by De Beurmann and Gougerot.[19] The causative agent is a yeast-like fungus of either the Schenkii or Beurmanni type. It multiplies by budding, and in cultures it forms a mycelium composed of freely branching septate hyphae, which in turn give rise to secondary conidia. The cutaneous lesions which develop in this condition are difficult to differentiate from the primary lesions of syphilis, cutaneous tuberculosis, and the ulceroglandular type of tularemia. The fungi of the sporothrix group are very sensitive to the iodides, and many of the reported cases have shown a complete cure following a short course of treatment. In the more resistant cutaneous lesions, unfiltered x-ray radiations are used in combination with the iodides.

Aspergillosis:[23-35] Aspergillosis is caused by mold-like fungus, possessing a basal stem and a stalk with a spore-bearing head. There are three varieties of aspergillus which may, under conditions of lowered pulmonary resistance, cause an infection of the lungs: A. fumigatus; A. Flavus; A. Niger. This infection appears to have a definite affinity for involvement of the pulmonary system. The local pathology is characterized by the appearance of granulomatous nodules on the mucosa, which runs a chronic course.[1]

In this country, aspergillus invasion does not seem always to be of the pathogenic type, and in a suspected case it is always a question of considerable discussion as to whether or not the fungus is purely a contaminant or a secondary invader.

In a public health survey of the iron mines in the Pitcher, Oklahoma, district, in which many chest roentgenograms were taken, Sayre and Merriweather[23] were puzzled to find in many plates, discrete, dense shot-like areas throughout both lung fields of workers who had never been ill.[23] From the sputum they were able to isolate the A. Fumigatus, Fisheri and A. Niger. Antigens from both of

these fungi were made, and when used, only the A. Niger cause a dermal reaction. When these plates were submitted to a group of eminent roentgenologists the diagnosis was as follows: (1) chronic miliary tuberculosis which has healed by calcification; (2) pneumonoconiosis; (3) pneumomycosis.

The condition has also been found among the wheat threshers of Oklahoma and Nebraska. Opie, in commenting on this particular condition, remarks that we are finding a type of inactive miliary tuberculosis, with increasing frequency. It is his opinion that a great many more cases recover than is believed.[23]

Occupation has a distinct relationship to the etiology of aspergillosis. It occurs most often in bird fanciers, pigeon breeders, hair sorters, millers, and in people who inhale dust, grain particles, and those who are constantly exposed to any form of dry or powdered vegetable matter. The onset of the infection is insidious in character, accompanied by cough, asthma, anorexia, and an evening temperature, and in the early stages is frequently treated as a chronic bronchitis. Later in the chronic course of the disease there is a loss of weight, night sweats, and hemoptysis.[19]

Symptoms: Hemoptysis is frequently the initial indication of onset. These patients seek the advice of the physician often in dire distress because they have been coughing and spitting blood. This may vary from tinged or blood-streaked sputum to rather massive hemorrhage. They may not appear acutely ill unless the hemorrhage has been sufficient to deplete the body. The temperature will probably register normal or possibly a slight elevation, most likely below 100 degrees. There may be slight dyspnea or breathing of an asthmatic type. Physical examination of the chest will probably reveal moist or sibilant rales about the hilus, or around the base of the lungs, while all the other physical signs will prove negative. The patient almost universally coughs up black granules resembling soot or coal dust which represent cast-off conidia.[18]

Fluoroscopic inspection of the chest in pulmonary aspergillosis will give very definite findings or possibly nothing on which one would or could hazard a diagnosis. A slightly increased infiltration in or about the hilus may be noted, and possibly a slight limitation of expansion of the chest may be present. The roentgen-ray findings are much more definite and conclusive. The absence of tuberculosis is readily established, although both diseases may be present in the same individual. Usually there is a marked thickening of the

bronchi leading to one or more lobes, while the adjacent lung fields show a rather marked infiltration with gradual fading out of the process in the more distant lung fields. In some cases the shadow may assume a cauliflower-like appearance. The picture may closely resemble bronchopneumonia in the early stage of resolution. The apparent well-being of the patient, along with the history, will rule out the possibility of pneumonia. Many eminent roentgenologists make the unqualified statement that this condition cannot be diagnosed from the roentgen-ray studies alone, but certainly roentgen-ray findings often point the way and make the differential diagnosis possible by eliminating other possible causes that might bring about such physical and clinical manifestations.[35]

Positive laboratory findings are in a sense essential for a definite diagnosis, especially if we are to complete the three-point diagnosis in reference to tuberculosis. It is a well known fact that molds are frequently found in mouths of apparently healthy individuals, and necessitates a thorough cleansing of the mouth before a specimen is obtained for examination. The bloody sputum is usually copious and may be almost entirely blood. It is usually free of tissue fragments or caseous material. In the hands of experienced bacteriologists, the aspergilli are readily recognized. Finding of these cells, however, does not clinch the diagnosis, but must be considered in conjunction with other findings.

Treatment: Early recognition of aspergillosis is essential to rational and effective treatment. If it is not recognized and goes along untreated it most probably becomes a chronic affair, resulting in multiple abscess formation, often proving intracticable or even fatal, although most cases will eventually recover. It is of very great importance that the differential diagnosis between tuberculosis and mycosis be established in order that rational therapy may be instituted. The iodides are specific in mycosis, while in tuberculosis they would prove disastrous. Potassium iodide may be given in full doses, and sodium iodide may be given intravenously to good advantage.[2] If this line of treatment is instituted early, the results are spectacular. Concomitant tuberculosis and mycotic infection of the lung contraindicates the use of iodides. Abscess formation with destruction of lung tissue is fortunately a rather slow process, and ample time is allowed for close study and a correct diagnosis before treatment is begun. In chronic cases small repeated doses of roentgen-ray is of great help, as is the case in practically all types of fungus infection. When abscess formation actually takes place it soon becomes a mixed infectious proposition with all of its serious consequences.

SUMMARY

1. Certain mycotic infections of the lungs have long been recognized as entities. Actinomycosis and blastomycosis are the common examples. Their known pathogenicity to man, and their ability to produce readily local lesions, and to cause a systemic invasion and visceral involvement, and eventual death, have established them as definite clinico-pathologic entities.[29]

2. Mycotic disease affecting the respiratory tract can be definitely diagnosed by bronchoscopic examination and study. Aspiration of exudate in the bronchi, uncontaminated by infections found in the mouth and pharynx, combined with bacteriologic study, is the certain method of recognizing these maladies in diseases of the pulmonary system.

3. Most cases of this nature or type are sent into tuberculosis sanatoria with suspected tuberculosis, and may be treated as such for a long period of time, or until the mycosis is finally diagnosed and proper therapy instituted.[19]

4. Positive diagnosis by clinical observation, repeated negative sputum for tubercle bacilli, the bronchoscopic aspiration, and the proper treatment other than that for tuberculosis, is important in mycotic disease, since in the mycoses the iodides offer the most hopeful method of cure, which in tuberculosis should not be used because of its detrimental effect.

5. Proper x-ray interpretation aids immensely in diagnosis.

6. Final diagnosis depends upon aspirating affected exudate through the bronchoscope, and developing cultures to differentiate between tuberculosis and mycosis.

7. Treatment, in mycotic infections, depends on organism isolation and is outlined in detail in the different subdivisions of this article.

8. A bronchoscopic department should be included in the clinical setup of all hospitals treating diseases of the broncho-pulmonary system.

REPORT OF CASES

CASE 1.—W. L. T., a white married male, aged 35, of American birth, who by occupation was a railroad fireman, entered the hospital complaining of a severe pain in the upper right quadrant of the abdomen.

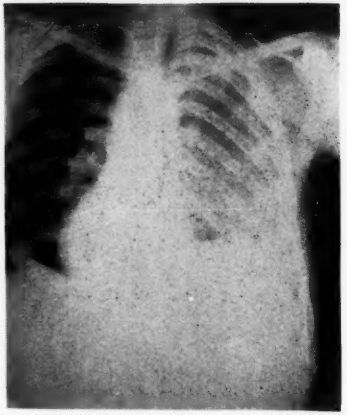

Fig. 10, Case 1. Pleural shading over entire right side increasing toward the base. Possible effusion. No cavitation.

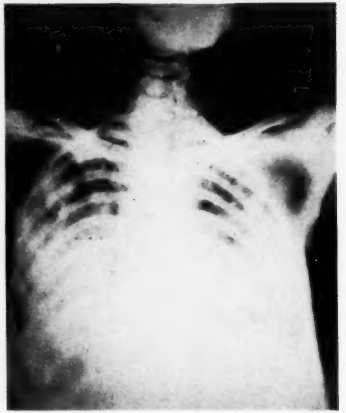

Fig. 11, Case 1. Pneumonic consolidation both lungs with multiple abscesses.

Fig. 12, Case 1. Pneumonic consolidation lower half of right lung, and multiple cavitation. Elevation of right side of diaphragm.

The family and past histories were negative except for cerebrospinal fever and influenza in 1918.

Present Illness: Since 1925 his health had been good, and he had worked as a railroad fireman until the past few months. About three weeks prior to admission (about the last of July, 1931) the patient awoke during the night with a severe pain in the epigastrium. He took an emetic which caused vomiting and was given a cathartic by a physician. He felt well again in a few days. No diagnosis was given him. After about a week the pain returned and continued. The pain was severe and almost constant in character, being increased in severity by intake of food. The only food tolerated was milk and water, which could be taken in small amounts. There was a slight fever but no recurrence of vomiting. The bowels were regular. There was no jaundice and no blood had appeared in the urine.

On admission, temperature was 99.6 degrees, pulse 88. The following evening, temperature rose to 101 degrees. Physical examination showed fairly well nourished white male, age 35, normal weight 168, but at present 155 pounds. The patient was acutely ill and suffering with severe pain in upper right quadrant of the abdomen, which was quite tender to touch. The cardiovascular system was normal; blood pressure was 118-86. The lungs were clear; the diaphragm normal in height and moved normally with respiration. Genitourinary and rectal examinations were negative. The abdomen was full, with a board-like rigidity in upper portion which was more marked in the upper right quadrant. The liver and spleen were not palpable. The blood count showed 16,800 leucocytes, with 80 per cent polymorphonuclear neutrophiles.

Treatment consisted of withholding food and giving saline 2000 cc. daily by rectum. Hot, wet fomentations were applied to the upper abdomen; narcotics and sedatives were given for relief of pain. The urine was negative and the chest x-ray showed nothing abnormal. The gall bladder examination gave the impression of disease of the gall bladder. The blood count was constantly rising, and on the 18th of August the leucocytes were 25,300 with 92 per cent polymorphonuclear neutrophiles. A provisional diagnosis of acute cholesystitis was made and an exploratory laparotomy was done the same day. The liver and gall bladder were found to be normal, although the liver was firmer to the touch than normal. Recent adhesions bound the anterior surface of the liver to the anterior abdominal wall near the midline. The adhesions were separated and about 50 cc. of pus escaped from a subphrenic type of abscess, ap-

Fig. 13, Case 1.

Fig. 14, Case 1.

Fig. 15, Case 1.

Fig. 13, Case 1. The lung is honeycombed throughout with small cavities ranging from 3 mm. to 1 5 cm. in size, and which are filled with a thick yellowish creamy pus. Fig. 14, Case 1. This lung is studded throughout with abscesses, practically no lung tissue remaining, and the little remaining between the abscesses is pneumonic and entirely consolidated. Careful examination of the contents of the abscesses in both lungs reveals that nearly all contain some small yellowish "sulphur granules." These, when crushed and stained by Gram, reveal a dense feltwork of mycelium, many of whose ends appear to be clubbed, though the radiate arrangement is not visible. Fig. 15, Case 1. Liver.—Centered about the gall bladder notch and fanning out from either side in large irregularly rounded lobules is a soft, necrotic, grayish mass, mottled with greenish streaks. It is totally necrotic, fairly firm, and pressure brings out a few areas of purulent softening and abscess formation throughout the entire liver substance. Without the brawny induration, this appears almost ilke an actinomycotic growth, with many small granules in the pus. Culture from this area shows the same organisms as noted in the lung.

parently located between the diaphragm and anterior surface of the liver. Drainage was established.

The postoperative course was one of gradual retrogression. The temperature was septic throughout, with periods of days when noticeable clinical improvement was apparent and temperature declining. Such apparent improvement, however, was not real. Leucocytosis was essentially a constant finding, varying from 13,000 with 85 per cent polymorphonuclear neutrophiles (three days postoperative) to 17,000 with 79 per cent polymorphonuclear neutrophiles one month later. In 60 days the leucocytes were 22,000 with 80 per cent polymorphonuclear neutrophiles. On December 18th, total count had risen to 30,000 with 81 per cent polymorphonuclear neutrophiles, and five days later had declined to 10,400 with 74 per cent polymorphonuclear neutrophiles.

An x-ray September 15, 1931, showed an elevation of the right diaphragm with thickened pleura and obliteration of costophrenic angle, suggestive of the presence of fluid. Sixty cubic centimeters of sterile fluid were removed by aspiration. On the 24th of September, another sixty cubic centimeters of slightly straw-colored fluid was removed from right chest. Pain in right lower quadrant increased, and exploratory laparotomy was done September 29, 1931, which revealed a small honeycomb-like abscess on the surface and within the substance of the liver. About two inches of the ninth rib, between the midaxillary and anterior axillary, was removed, and tube drainage instituted. Abdominal distention became a distressing symptom for the following four or five days. Relief was obtained by repeated compound oil enemas with pituitrin intramuscularly. X-ray examination was negative for intestinal obstruction and rectal examination was negative. On October 20, 1931, a 3 cm. incision was made where, with evacuation of 150 cc. of frank pus, the cavity did not seem to penetrate further than the muscular layers, and healed satisfactorily under Dakins irrigations. Cultures showed staphylococcus albus and streptococcus, nonhemolytic.

On November 3rd, x-ray reported fluid at the right base. On November 27th, it was noticed that while irrigating with Dakins, through rib resection wound, paroxysms of coughing were produced, and patient stated that he could taste the solution. Gentian violet was placed in the wound to demonstrate a possible fistula but the patient did not spit up any of the dye. On December 17th, the procedure was repeated, and patient coughed up some of the dye, showing a communication between the pleura and the peritoneal

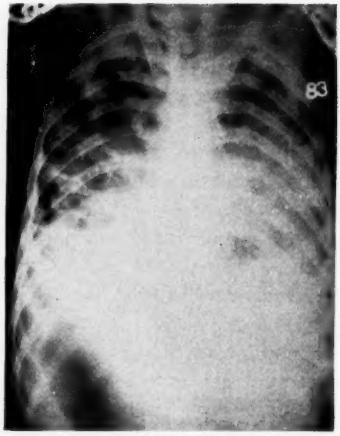

Fig. 16, Case 1. Right lung shows uneven nodose mottling, and is indicative of multiple lung abscesses. Left lung shows indefinitely outlined multiple cavitation throughout. Small area of pneumothorax is seen extending downward from the level of the 4th rib in the anterior axillary line. Towards the medial half of the lung, behind the 3rd interspace, and lower border of the 4th rib, anteriorly, are seen two fluid levels. The appearance of both lungs is that of multiple absceas cavities.

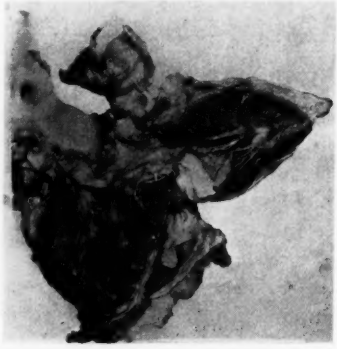

Fig. 17, Case 3. In the myocardium of all chambers there are numerous large and small abscesses containing thick, greenish-yellow pus. These appear at all levels and involve some of the columnae carnae and papillary muscles.

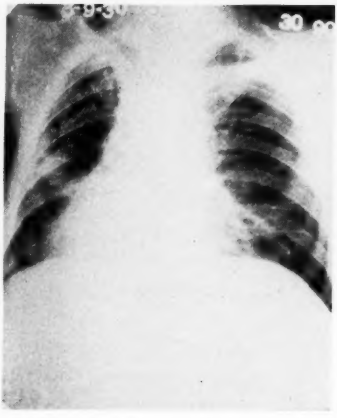

Fig. 18, Case 4. Bilateral involvement. Multiple small cavities in right base.

cavity. On the night of the 17th, the pulse became very thready and rapid, and the heart considerably dilated. Digitalization by massive dosage was instituted, with noticeable improvement in thirty-six hours. On the 29th, x-ray showed more extensive involvment within both lungs, bronchopneumonia in type, and the possibility of early, multiple, pulmonary abscesses was considered. Rales present in both bases, posteriorly. Bronchovesicular breathing, anteriorly, on the right. Oxygen given continuously. Diathermy on both sides of the chest daily.

X-ray of chest, December 21, 1931: A single bedside examination in supine position showed that there was a definite clearing of the thickened pleura over the right chest, but throughout this lung there were present large opaque, rounded and oval shaped opacities, extending from between the second rib to the base, which were not present in the previous films. The left lung, which at the previous examination was rather clear, at this time from the apex to the base were scattered through it varying-sized opaque areas mostly oval in shape, and considerable reaction appeared present through both lungs. Findings in both lungs were now believed indicative of multiple abscesses. The trachea at this time lay more to the right than previously, as also does the cardiac shadow.

X-ray of chest, January 7, 1932, based on a single bedside examination with patient lying down, showed a definite increase in the amount of clouding through both lungs, more so in the left than the right. The clouding in the left lung below the third rib level was almost uniform. Above that level there was more uneven density, and as a whole was definitely less dense. Some fluid was thought to be present in this lung. There was a definite increase in the pneumonic reaction throughout this lung. The right lung showed a heavier mottled opacity, which was believed to be indicative of increased reaction in the lung, apparently around multiple abscesses. The right leaf of diaphragm showed a rounder dome, but it also rose higher at this time. Compared with previous examination, there was a definite retrogression in the process in both lungs at this time.

On January 5th, 575 cc. of clear straw-colored fluid was aspirated from left chest, posteriorly, and repeated one week later, when 150 cc. of fluid was obtained. Cultures of this fluid were negative. Patient retrogressed rapidly, showing extreme emaciation and edema of both lower extremities. Pulse, however, remained fairly good. X-ray on the 15th showed multiple multilocular cavities throughout both lungs.

Patient died at 11:00 a. m. January 17, 1932.

In addition to the procedures outlined above, patient had been given rather massive doses of siomine (an iodine preparation) with apparent benefit.

X-ray of chest, January 20, 1932, which was based on a single postmortem examination, showed the pleura thickened over both lungs. Trachea was pulled slightly to the right. The right leaf of the diaphragm appeared to be held slightly high at its anterior side. The left leaf of the diaphragm was obscured by fluid in the base of the left chest. Two fluid levels are shown, one apparently just above the diaphragm and one about one rib and interspace higher. Right lung showed through it an uneven, nodose mottling of less degree than at the previous examinations, but this mottling, although somewhat bronchogenic in type, was believed to be indicative of multiple lung abscesses. Left lung showed indefinitely outlined multiple cavitation throughout. Pneumothorax extending downward from the level of the fourth rib in the anterior axillary line was revealed. Toward the mesial half of the lung, behind the third interspace and lower border of the fourth rib, anteriorly, were two shadows somewhat suggestive of fluid levels. Compared with previous examination there had been considerable clearing of the uneven clouding through both lungs.

Laboratory Findings: In addition to the above, guinea pig inoculation of fluid from right chest was negative for tuberculosis, and repeated blood cultures were negative.

Blood Chemistry: Showed sugar 86, nonprotein nitrogen. 42. Urine was repeatedly positive for albumin since December 6, 1931.

Clinical Diagnosis: (1) Subphrenic abscess right; (2) pleural effusion, right, secondary to No. 1 (staphylococcus albus); (3) liver abscess; (4) pleurisy chronic fibrinous, bilateral; (5) abscess, abdominal (streptococcus, nonhemolytic); (6) empyema, right; (7) bronchopneumonia, bilateral; (8) lung abscess, multiple bilateral; (9) peritoneo-bronchial fistula.

Autopsy—Lungs: The entire surfaces of both lungs are dotted over with small pleural and subpleural abscesses, giving the surface a coarsely honeycomb appearance. They were easily broken, and exuded pus on pressure. The abscesses were more numerous subpleurally on the left and contained thick yellow pus. The right lung was honeycomb throughout with small cavities ranging from

3 mm. to 1.5 cm. in size, and were filled with a thick, yellowish, creamy pus. The diaphragm was densely adherent to the liver, and to the lower lobe of the right lung, and at its tip there was a perforation through from the abscess between the liver and diaphragm into the pleural space. The left lung corresponded to the right in most details, with the exception of the empyema noted above, which occupied the antero-lateral surface of the lower lobe, with fibrinous purulent pleurisy over all this area. This lung was studded practically throughout with the abscesses, almost no lung tissue remaining, and the remaining between the abscesses was pneumonic and entirely consolidated. The congestion was not so marked as one might expect. In practically all of this exudate there was found a pneumococcus and a Gram positive, slender bacillus, slightly beaded, which resembled the tubercle bacillus stained by the Gram method. Careful examination of the contents of the abscesses in both lungs revealed that nearly all contain some yellowish "sulphur granules." These, when crushed and stained by Gram, revealed a dense feltwork of mycelium, many of whose ends appear to be clubbed, though the radiate arrangement is not visible.

The Liver: Over the right lobe of the liver the diaphragm was densely adherent, and at the midportion of the right lobe was an area in which the original liver abscess was opened, and where the perforation of the diaphragm occurred. Between the liver and the diaphragm, a small abscess cavity was found with sinuses leading upward to the drained wound in the eighth interspace and downward to an abscess cavity in the right lobe. The liver surface, except for the area of dense adhesion, showed slightly thick fibrinous deposit. Centered about the gall bladder notch and fanning out from either side were large irregularly rounded lobules of a soft, necrotic, grayish mass, mottled with greenish streaks. It is totally necrotic, fairly firm, and pressure brings out a few areas of purulent softening and abscess formation throughout the entire liver substance. Without the brawny induration, this appears almost like an actinomycotic growth with many small granules in the pus. Culture from this area showed the same organisms as noted in the lung. In addition, a few of these granules reveal the same picture of Gram stain as described for those in the lung abscess. Many minute abscesses were noted throughout the entire liver, but are centered about the gall bladder. The gall bladder, however, showed the ducts patent with no stones, and the gall bladder itself was not changed.

Anatomical Diagnosis: (1) Actinomycosis both lungs, with multiple abscesses throughout all lobes and in pleura; (2) actino-

mycosis of liver, with multiple abscess formation, incision, drainage, sinus formation over right lobe, subphrenic abscess, perforation through diaphragm into right pleura; (3) empyema left (530 cc.); (4) pleurisy chronic fibrous adhesive over all lobes bilateral; (5) diaphragmatic and pericardial adhesions bilateral; (6) dilatation cardiac right terminal; (7) splenitis chronic toxic; (8) nephritis parenchymatous toxic.

Liver (microscopic examination): All sections revealed a widely distributed interlobar fibrous overgrowth in which were many compressed bile ducts. About the abscesses was an especially heavy fibrosis. The abscesses themselves showed many fine strands of what appears to be fibrous connective tissue ramifying throughout the pus in a sort of supporting reticulum, which accounts for the non-fluid nature of the necrotic abscess contents. Many irregular colonies of actinomycosis are found in the various abscesses.

Resume: As is evident in this case of severe infection with actinomycosis, it often resembles tuberculosis, and is probably often mistaken for it. The characteristic thing about most of this type of infection is the disproportion between the symptoms and the amount of pathological change. The cough is usually only moderately severe, without a large amount of expectoration; the fever is of a low type, the leucocytic ranged higher, due to liver abscess and lung abscesses.

Actinomycosis is pathogenic in the lungs, and is invariably fatal. In this case there was liver pathology with abscesses which showed the characteristic tendency on the part of the disease to burrow its way in all directions, toward liver, pleura, and peripleural tissues, with erosion of the ribs, and the formation of a subphrenic abscess.

Case 2.—S. M., a white male, aged 50 years, of American birth, and married, who by occupation was an attorney, was admitted to the hospital with the complaint of sciatica and nephritis.

Previous history negative; he was an abstainer and gave no history of venereal infection.

While in France, in 1918, patient developed nephritis, which was attributed to exposure and an almost entirely meat diet. Was hospitalized there, and later sent to the United States, and discharged with disability due to interstitial nephritis. There were periods of illness off and on for the next five years, and at times he was confined to bed with these attacks, which were due to an exacerbation

of his nephritis. There were frequent headaches and backaches, the urine was scanty and attacks of nausea were frequent.

In 1925 the patient had an initial attack of severe sciatica, right side. He was admitted to the hospital for treatment of sciatica and nephritis. His tonsils were removed, and he subsequently developed a lung abscess in the middle lobe of the right lung with an associated pneumonia.

Recovery and general improvement followed, but cough and profuse expectoration continued, which along with his nephritis showed occasional exacerbations.

He was discharged from the hospital but failed to regain his strength and the symptoms persisted. He was readmitted in 1930, complaining of weakness, cough, and expectoration of foul material. He thought the urine was blood tinged. At times he vomited and always felt better afterwards. There was some chest pain, mostly under the right side, and he coughed up blood, usually small amounts, but about one year previous brought up quite a bit. Temperature was usually subnormal, but there had been occasional elevation. Occasionally night sweats occurred and there was dyspnea on least exertion. A nasal discharge was present and he complained of headache and occasional spells of frequency and voiding of large amounts of urine, which often appeared bloody. There were aches and pains in the various joints, especially in the left hip.

Physical examination revealed a well developed and fairly well nourished male; not acutely ill; the respiration was rapid and dyspneic. The pulse and temperature were normal. The skin, mucous membranes, glandular system, osseous system, heart, abdomen and nervous system showed no deviations from normal. Examination of the lungs revealed the presence of an abscess in the lower right lobe.

Progress of Case: The patient's condition was one of retrogression. At times he was very sick, showed signs of toxemia. The cough was excessive and produced a great deal of foul sputum. Postural drainage was used three times a day. He was bronchoscoped January 9, 1931, and several times since, and a moderate amount of pus aspirated. The temperature varied from 98 to 101 degrees almost daily. The pulse was rapid and there was a leucocytosis of 26,000; chills and sweats were frequent. Sputum was reported negative for tubercle bacilli.

X-ray of chest, January 22, 1931: The whole right base shows an area of pneumonitis which extends upward beyond the third rib. Throughout this area there were multiple areas of rarefaction, showing fluid levels. In the lateral film there is seen a cavity showing a fluid level. The cavity is about 5 cm. from the skin, and is about 5 cm. from top to bottom in depth, extending from the second interspace downward; multilocular cavitation is present in the lung tissue.

In January, 1931, a crucial incision was made over a joint of maximum swelling in the region of the third rib anteriorly. Necrotic muscle tissue was exposed and a sinus tract was found between the third and fourth ribs. Portions of the third and fourth ribs were removed. The pleura was opened, and three abscess cavities in the lung were found, which were drained and packed with vaseline and iodoform gauze. Smears of pus and degenerated tissue removed at operation showed many fine mycelial threads, apparently a member of the nocardia. No growth occurred on ordinary culture media, or on Saborund's media. Patient was given scomine, five grains a day for a month. This is an iodide preparation, but as it upset his stomach, was discontinued. The wound in his chest was packed with a solution of ½ per cent iodine and 10 per cent K.I., which cleared up his abscess and sinus tract.

In March he gained a little weight; cough and sputum lessened. The sputum was not reported negative for tuberculosis but positive for actinomycosis.

In June, patient developed a generalized hemolytic streptococcus infection, and due to myocardial involvement he failed rapidly and died.

No autopsy was performed in this case, and clinical diagnosis consisted of: (1) Abscess, pulmonary, lower lobes, due to fungus infection (actinomycosis); (2) thoracotomy, anterior ends of third and fourth ribs, with evacuation of pus in pulmonary and superficial tissue abscesses; (3) septicemia, due to streptococcus hemolyticus.

Case 3.—N. W., a 43-year-old soldier, married eight years, and had tropical service (in 1916) in Mexico. The ages and causes of death of mother, father, two brothers and one sister were unknown, two daughters having died of pneumonia, but wife and other child were well. There was no history of tuberculosis or cancer in the family; the patient denied use of alcohol and drugs. His childhood diseases were measles and mumps; in 1923 pneumonia; and

in 1924 had excision of enlarged glands in supraclavicular region, right.

Present Illness: Patient stated he first had pain in chest in July, 1936. At that time he had a dull aching pain, non-radiating in character, near left lower costal margin, in midclavicular line. This pain became more frequent and annoying. In the latter part of July the pain was noted over lower sternum. Between the middle of October and the middle of November, 1936, he was practically symptom-free, but shortly thereafter he noticed a swelling in left pectoral region, which became more marked about November 18th. The swelling was rapid, and pain was increasingly severe. The swelling ceased after about one week. He was admitted to the hospital on December 21, 1936. On admission he was found to be ambulant, fairly well nourished, and on the anterior portion of the chest was found a draining sinus of thin yellow purulent material. Eyes, ears and nose were normal. The skin was dry and scaling over left anterior of chest. Blood pressure was 120/60, pulse 109 sitting. Heart had not enlarged to inspection.

Patient's course was one of continual regression. Multiple superficial soft elevated nodules (possible fungus infection) appeared over forehead and extremities. Blood culture was negative repeatedly. The discharge of these nodules was thin and dark brown in color. On March 25, 1937, a nodule first appeared over the left paretid area. This was incised and it eventually healed. In April a shower of nodules rapidly appeared there again. Several were aspirated, still he slowly regressed. Regression continued until death occurred at 6:20 a. m. May 5, 1937.

Laboratory Findings: W. B. C. 13,750; lymphocytes 18, monocytes 4; neutrophiles 76; eosinophiles 2. Wassermann negative. Specimen from abscess of jaw, the smear showed many pus cells, no organisms were seen; cultures showed no growth; blood culture showed no growth. E.K.G. slight sinus arrhythmia. Slight abnormality of some of the principal deflections of Q.R.S. Slight sinus tachycardia. No definite evidence of cardiac pathology.

Clinical Diagnosis: (1) Tuberculosis, pulmonary, chronic, active, all lobes left lung; (2) pleurisy, chronic, fibrous, tuberculous, left; (3) spondylitis, chronic, tuberculous, involving fifth, sixth and seventh thoracic vertebrae; (4) abscess, chronic, tuberculous, posterior mediastinum and anterior chest wall, left pectoral region; (5) arteriosclerosis, generalized, moderately severe, cause undetermined; (6) aortitis, chronic, ascending aorta, moderate, cause un-

determined; (7) abscessed, multiple, superficial, soft, all four extremities and scalp, cause undetermined. No tubercle bacilli or fungi found antimortem.

Lungs (autopsy): In the apex and throughout the upper lobe are felt numerous shotty nodules. A few similar nodules are felt in the middle and lower lobes. On section, the lower lobe is much darker red than the upper and middle lobes, and very bloody, but there is no definite consolidation. The upper and middle lobes, except for the small firm nodules, are relatively normal in appearance. Left lung: the anterior border of the upper lobe is tightly adherent to the pericardium, to form a mass in this area, which is firm and on section in this mass numerous areas of suppuration are found. In this area the lung parenchyma is very firm and relatively airless. Small grayish-yellow nodules are seen in the lung parenchyma. On section there are found numerous small, thin-walled cavities in the apex, and several shotty nodules throughout the remainder of the lung. On the lower anterior aspect of the upper lobe is a large yellowish ragged area which is directly beneath one of the superficial abscesses of the chest wall.

Liver (autopsy): Through the capsule are seen numerous irregularly shaped yellowish lesions which on section appear to be abscesses in the parenchyma just beneath the capsule. On section there are a few similar lesions in the liver parenchyma.

Spleen is small. On section an abscess is seen. Several rounded, soft, yellowish nodules are seen in the parenchyma. On section some of these are firm, and others contain yellow pus.

Pancreas appears enlarged, and on section are found numerous abscesses in the parenchyma.

G.I. Tract: Numerous small abscesses seen in gut wall. All contain thick yellow pus.

Heart: In the myocardium of all the chambers there are numerous large and small abscesses containing thick, greenish-yellow pus. They involve some of the columnae carnae and papillary muscles. Similar abscesses were also found in the thyroid, mediastinal lymph nodes, kidneys and left testicle.

Cause of Death (anatomical): (1) Actinomycosis, involving subcutaneous tissues, thyroid gland, mediastinal lymph nodes, pericardium, myocardium, endocardium, liver, spleen, pancreas, kidneys, left testicle, small and large intestines and mesenteric nodes; (2)

tuberculosis, pulmonary, chronic, inactive, all lobes both lungs; (3) bacteria, tuberculous, without tubercle formation. (Note: Tubercle bacilli demonstrated by smear and culture from suppurating lesions of pancreas and heart and subcutaneous tissue. Histologic study reveals typical actinomycosis lesions and no tubercles.)

Resume: The patient had an old healed tuberculosis pulmonary infection in his infancy; later, he developed an actinomycosis, which lowered his resistance so that by erosion it was able to break off the healed tubercle node or lesion causing a miliary tuberculosis and death.

CASE 4.—S. J., a white male, a jeweler, single, aged 42, born in Poland, came to the United States in 1910. He used alcohol and tobacco moderately. Family history was essentially negative. Previous history: rheumatism at age of 21; influenza while in the Army. Denies venereal infection.

Present Illness: One and one-half years ago patient began to feel bad. Since 1923 he had been in a run-down condition. In August, 1929, he had a "cold," and following this there were some gastric disturbances. In September, 1929, he went to another hospital, suffering from pneumonia. He continued to feel badly until he entered the present hospital on December 17, 1929.

The gastric disturbance had been characterized by pyrosis, most severe in the afternoons. Later he developed eructations of gas, flatulence, regurgitation, bad taste in mouth, chronic constipation and heartburn. His appetite was poor, but had no jaundice or abnormal pain, with the exception of a few indefinite chest pains. Other symptoms were chills at varying intervals since November, 1929, moderate dry cough, frequency of urination, and rarely hematuria.

On admission to hospital his general condition was poor. There was present a marked weakness, a sallow color, underweight 14 pounds, a somewhat toxic appearance, a blood pressure of 86/64, a slightly enlarged heart, systolic murmurs at apex, an enlarged and dense spleen, clubbed fingers. At first rales were heard, through third rib, posteriorly over left chest, anteriorly over left apex. On December 28, 1929, the liver was found to be enlarged moderately and slightly tender. He was diagnosed liver abscess. Patient continued to have irregular fever with chills and sweats. Tenderness persisted in right upper quadrant. In March, patient was semi-ambulant. Had some cough and expectoration, pain and tenderness

below right costal margin, backache in right kidney region. Blood pressure 110/72. He was constipated, had clubbed nails. The liver abscess was opened and drained of about one liter of pus. Following this, the left tonsil became swollen, and there was some swelling of the glands of the jaw. On May 5th, left ear drained considerably. Erysipelas developed about this area. June 24th he had a moderate cough, slight expectoration, marked clubbing of the nails, stubborn constipation, and his heart enlarged, with soft systolic murmur at apex. On September 15, 1930, patient had feeble breath sounds, diminished resonance and rales over the left base. The examiner suspected pneumonia of the left base. September 30, 1939, blood pressure 76/58, pulse weak and irregular. Heart sounds weak. At this time he had a white blood count of 20,250, polymorphonuclear neutrophiles 82 per cent. Temperature 104.4 degrees.

On October 1st it was thought that the liver abscess had recurred. A marked tenderness was found over the epigastrium. On October 2, 1930, patient was again operated on for liver abscess. Moderate amount of pus obtained from dome of liver. Multiple abscesses were still suspected. The wound was packed with iodoform gauze and rubber drainage tubes were inserted. Following the operation a moderate amount of pus drained daily. On October 13, 1930, patient had some hemoptycosis, but no frank hemorrhage. On October 17th, a large amount of extremely foul smelling grayish pus drained from the wound. Patient's temperature, which had previously been high, dropped to normal limits. About November 6, 1930, jaundice increased. Twenty per cent glucose in 250 cc. amounts were given daily. The patient's condition grew gradually worse. On November 9, 1930, the blood pressure was 84/40.

On November 13, 1930, at noon, the patient was somewhat cyanotic. Oxygen was given by the nasal tube. The respirations 50-60, pulse was thready in character, and the lungs were clear. The patient expired at 3:45 p. m. Death was listed as cardiac.

Laboratory Report: X-rays, December 21, 1929: Thorax shows marked contraction of both uppers. Heart enlarged to left. Thickened pleura over left lung. Marked fibrosis through left lung. Apparent multiple cavitation above fourth rib.

X-rays, October 11, 1940: Left lower lobe denser. Left leaf of diaphragm obscured by shadow.

Urine: Negative until April, 1930, at this time albumin was positive, and occasional hyaline and granular casts were found.

Stools: Occult blood and free fat throughout January, 1930.

Sputum: Persistently negative for tuberculosis.

Blood picture: On admission, 4,500,000 R. B. C., 6,550 W. B. C., hemoglobin 60 per cent, polymorphonuclear neutrophiles 60 per cent. December 31, 1929, 19,700 W. B. C., polymorphonuclear neutrophiles 86 per cent. November 1, 1930, 31,900 W. B. C., polymorphonuclear neutrophiles 91 per cent, hemoglobin 50 per cent. The W. B. C. was high throughout the stay in the hospital.

Blood chemistry: December 18, 1929: Sugar 97.5 E.P.E. 31.57. October 1, 1930: Icterus index 90 Vandenbergh prompt positive.

Diagnosis: (1) Tuberculosis, pulmonary chronic, active, all lobes, both lungs, fibrous type, scattered minimal nodose fibrosis throughout, with fibrous interlobar pleurisy between upper and middle lobes; left lung marked fibrous adhesions with irregular cavitation above fourth rib to base; dense area of fibrosis in periphery between third and fourth ribs; (2) pleurisy chronic fibrous bilateral (?); (3) multiple abscess of liver.

Autopsy—Lungs: The right lung is voluminous and covered with thickened pleura. It is normally crepitant throughout. It shows edema and congestion of the lower lobe, more prominent in the posterior portion (hypostatic). The upper lobe is fibrous at the apex and presents a single old calcified and fibrous lesion. The left lung is also voluminous. It is removed within the parietal pleura because of the dense adhesions. Over the small encysted empyema on the lateral surface of the lower lobe, the pleura is especially thick, measuring about 5 mm. The pus in this sac is thick, creamy, yellowish and smooth, and shows no globular masses, as far as the eye can detect. Over the upper lobe there are several scattered nodules about 1 cm. in diameter, which are easily palpable beneath the pleura. The base of the lower lobe is solid, airless, and feels as if it were the seat of a bronchopneumonic consolidation. On section, the upper lobe is for the most part quite normal in appearance, but is slightly edematous. The subpleural nodules above mentioned prove to be small abscesses with thick yellowish walls, filled with a viscid pus much the same as that in the encysted empyema. The lesions in the lower lobe are rather unusual in type. They have the conglomerate, lobulated appearance of bronchopneumonia in the later stages, but they do not surround or apparently proceed from bronchi. They have a distinctly tan color, and seem to be encapsulated. The majority are firm in consistency, but a few have become softened and

Fig. 19, Case 4. Involvement of both lungs with multiple cavitation on right side.

Fig. 20, Case 6. Improvement in pleural thickening. Increase in parenchymal involvement.

Fig. 21, Case 6. Deformity of right side of diaphragm by adhesions. Marked thickening of pleura over base of right lung, and also interlobar pleura. Parenchymal infiltration.

necrotic at the center. The general appearance of this group of lesions is that of a more or less irregular center from which have spread numerous rounded, lobulated, secondary extensions. There is a single isolated lesion of this type, with a softened center, in the upper portion of this lobe. Dense, tough, fibrous adhesions bind the base of the lung to the diaphragm and to the pericardium. The lower margin of the encysted empyema extends down to the dome of the diaphragm, and at this point the adhesions are much thicker than elsewhere.

Autopsy—Liver: Sections at various levels show multiple abscesses, six in number, varying from 1.5 to 5 cm. in diameter, rounded, filled with fetid, yellowish-gray, thick homogenous pus. The walls of these abscesses are heavy and fibrous and the parenchyma adjacent to them seems infiltrated by fibrous tissue to some extent. The lining of the abscess cavities is ragged, shaggy and gray. The abscess cavity drained by the operative wound was apparently of this type, and near the lower margin of the liver, above the gall bladder, it has contracted and shows beginning healing. Separate from these abscesses in location and type are several groups of lesions which more or less resemble those in the lower lobe of the left lung. They are irregularly rounded, lobulated, yellowish in color and, like those pulmonary lesions, seem to have extended in rounded masses from a common center. Each area is outlined by a light yellowish-brown zone with a darker center, is firmer than the surrounding parenchyma, appears to be encapsulated, and shows no central necrosis. Neither these nor the abscess cavities bear any visible anatomical relation to any of the liver structures. The parenchyma is soft, flabby and friable, is pale brown in color, and has a greasy feeling. With the opening and evacuation of the abscess cavities the tenseness of the surface disappears, and the prominence of the dome subsides. The lobular structure is obscured, and there is nowhere any tendency toward congestion, not even about the abscess cavities.

Autopsy—Spleen: The spleen is also greatly hypertrophied. On section, one notes a subcapsular abscess cavity beneath the diaphragmatic adhesion, about 1 cm. in diameter, filled with creamy, grayish-yellow pus, and thick-walled. Another lesion nearer the center is 2.5 cm. wide, yellowish, firm at the periphery, but soft with beginning necrosis at the center. The outline is irregularly rounded and somewhat lobulated. Two other similar but smaller lesions are seen above this one. The cut surface of the spleen is pale and somewhat cloudy, the Malpighian bodies and the trabeculae are obscured and the texture fairly normal.

Smears are made from the lesions of the left lung, the left pleura, the liver and the spleen. A smear from one of the solid areas in the lung shows no organisms of any kind. Smears from others of these lesions and from all other lesions show a Gram positive, beaded, branching mycelia, but no other organisms. Where the smears have not destroyed the structure of these groups of mycelial threads there is a suggestion of a radiate arrangement of the periphery, but no clubbing of the ends is noted. Cultures taken at the same time, and planted on various media, show no growth in 96 hours, either by aerobic or anaerobic incubation.

Findings (autopsy): (1) Pleurisy chronic fibrous adhesive bilateral; (2) actinomycosis with primary lesions in left lung both lobes; secondary mycotic encysted empyema left; mycotic abscesses in liver and spleen, and encysted between spleen and diaphragm.

Microscopical Examination—Lung: Sections from abscesses and from other areas of softening exhibit about the same picture. There is a uniformly increased fibrosis about them, either in the form of a definite capsule or of a more diffused process. Some areas are characterized mainly by a general thickening of the alveolar walls in the neighborhood of the suppurative foci. In the foci of suppuration and softening one sees frequent small lobulated bluish staining masses in the pus. These masses are homogeneous and seem structureless by ordinary staining methods, but by modified Gram stains and Mallory's eosin-methyl violet method, the mycelial structure of these colonies is brought out and also the peripheral zone of radiating hyaline mycelial thickening or clubbing characteristic of actinomyces.

Liver: Sections are made from the walls of the large abscesses and from the more fibrous lesions. In the sections from the large abscess wall one finds little but the picture of a very thick fibrous wall containing a small amount of pus and debris. Most of the abscess contents have escaped, and one sees none of the actinomyces colonies such as are found in the minute, unruptured lung abscess. The immersion lens reveals many small colonies and isolated threads such as were seen in the Gram stains of the pus smears from all of the liver lesions. Here in the liver one sees also, as in the lung lesions, the excessive fibrosis about and near these abscesses, corresponding in a less degree to the "brawny" induration so characteristic of actinomycoses clinically. Apart from these mycotic lesions there is a quite general interlobular fibrosis of a slight grade, and a general fatty degeneration.

Resume: Smears are taken from all lung lesions, from the encysted empyema, from the splenic and liver lesions. All these smears, except one from an old fibrous lesion in the lung, show the same picture (actinomycosis). Gram stains reveal many scattered mycelial filaments with frequent agglomerations of mycelium into small round colonies without clubs. No clubs are seen in any of the isolated mycelia, though it is to be noted that no special stain was made for them.

CASE 5.—M. C. G., Porto Rican, aged 20 years, a clerk, was admitted to the hospital January 20, 1923, from the Panama Canal Zone, with a diagnosis of tuberculosis pulmonary, acute, right upper lobe.

The family history was negative. Patient had tonsillitis one year before. Denies any other illnesses.

Present illness began in January, 1922, starting with pain in the right side of the chest, followed by a persistent cough and no expectoration. Although he had some fever and a cough, his general condition remained normal. The physical examination of his chest showed many crepitant rales over the right upper lobe, both anteriorly and posteriorly. X-ray showed a progressive involvement of the right lobe. He continued to lose weight. The temperature ran from 99.6 to 101 degrees, and he had a hacking cough. Sputum examination for tubercle bacilli was negative.

On February 6, 1923, he continued his coughing, some blood spitting, but experienced no pain in the chest, and no night sweats. He was diagnosed as tuberculosis pulmonary, chronic, active, all lobes, right lung.

In March, 1923, he was additionally diagnosed as a case of pleurisy sero-fibrinous right. At that time he had considerable elevation of temperature. Fluid was obtained from his right chest, which was found sterile and a "rapid spread of bronchopneumonia" was added to his diagnosis.

In April, his pneumonic process improved and his temperature remained normal for two weeks.

In May, 1923, he was diagnosed: (1) Tuberculosis, pulmonary chronic active right upper and lower lobes, inactive all other lobes both lungs; lesions, dense pneumonic, right upper; discrete nodose all other lobes both lungs; moderate perifocal reaction right upper and lower; (2) pleurisy sero-fibrinous, slight, right.

During this interval, the patient had daily elevation of temperature, persistently negative sputum for tubercle bacilli. His condition began to retrogress. He had moderate elevation of temperature, pulmonary abscess right lower lobe. The patient had had two negative Wassermanns, he was treated with neosalvarsan, and although he showed severe clinical reaction to his disease he tolerated the neosalvarsan well and patient improved.

In August he continued to have elevation of temperature. Twenty-six sputum examinations were negative for tubercle bacilli, the foul sputum showed only pneumococci and pyogens. A pneumothorax was done to collapse the lung abscess.

On September 8th a rib resection was performed to evacuate the pulmonary abscess, but his condition began to retrogress and patient died September 25, 1923.

The laboratory findings were: Wassermann negative. Stools negative. Urine was negative up to September, when casts were found. Sputums (60) negative for tubercle bacilli.

Clinical Diagnosis: (1) Actinomycosis pulmonary; (2) pulmonary abscess right upper lobe; (3) pleuritic adhesions right base; (4) pneumothorax artificial right.

Pleura and Lungs (autopsy): On the left the pleural cavity was practically normal. The lung was small but of normal pink color throughout, and crepitant. There was not even any hyperstatic congestion. There were no lesions present. Practically no pneumonoconiosis. On the right the lung was entirely collapsed. The lower portion of the cavity was filled with thin greenish foul pus, the midportion was adherent, and there was another pocket of pus in the upper portion with the apex firmly adherent. A sinus went into the lung as well. On sectioning the lung, the upper portion was a honeycombed mass of openings averaging about 1 cm. in diameter, with deep red granulating edges. Between the holes the trabicula were well fibrosed. In these fibrous strands were areas of yellow pepper grain pigment. This looked much the same as "autumn leaf" heart. Gross inspection does not reveal their true nature. In the lower portion of the lung there were grayish areas of small size which were breaking down and would probably eventuate in abscesses such as those in the upper portion. There were no tubercles seen anywhere in either lung. The left lung was practically normal. The right was apparently devoid of normal alveolar tissues.

The glands were enlarged, due to lymphadenitis from chronic infection.

Resume: This is another case of mycotic infection which suggested primarily a tuberculosis pulmonary, the sputum examination remaining constantly free of tubercle bacilli, but whose clinical course was one of actinomycosis.

CASE 6.—J. F. R., white American, aged 19, was admitted to the hospital February 26, 1936.

The past history showed that he had the usual childhood diseases. Denies venereal infection. He was a moderate user of alcohol. Mother died in 1925 of cause unknown. Father, four brothers and one sister are living and well.

On admission the patient gave a history of being sick on October 17, 1935, with a sore throat, headache, a cold and a fever. The blood count showed W. B. C. 7,950 and 69 per cent polymorphonuclear neutrophiles. Followed an attack of sudden pain in right chest, quite severe for an hour, he was not dyspneic, did not cough. His fever varied from 99 to 101 degrees. He had no symptoms referable to the cardiovascular system. No genito-urinary disturbances.

X-ray of the chest revealed evidence of right lung pulmonary tuberculosis of some chronicity and he was diagnosed: Tuberculosis, pulmonary, active, upper lobes right lung; pleurisy with effusion right, secondary to No. 1. Sputum examinations (22) were negative for presence of tubercle bacilli. Patient had lost 9 pounds in weight.

In February, 1937, the diagnosis was changed to pneumonitis, chronic, lower lobe right lung, caused by fungus (torula histolytica), since the mycotic cells, identified as torula histolytica, were found in the sputum; pleurisy, chronic, right, secondary to No. 1, and adenitis, chronic, suppurative, cervical, bilateral, caused by fungus (torula histolytica). The group of cervical lymph glands on the right side was removed by biopsy, and torula histolytica were found.

The laboratory findings were: Urine and feces, all negative; sputums (42) negative for tubercle bacilli. In May, 1936, sputum shows torula histolytica. Blood counts, W. B. C. 15,000, with lymphocytes, 26, monocytes 3, polymorphonuclear neutrophiles 71. Three months later, W. B. C. 14,650, and polymorphonuclear neutrophiles 74. Shortly after, patient's counts became normal and

temperature subsided. Wassermann and Kahn tests in both the blood and spinal fluids were normal. The tubercle bacilli were not found in the glands, and guinea pig inoculations from the glands were negative. Tuberculin reaction (Mantoux) 1/100 was very faintly positive. Blood chemistry, sugars 103, lipoids 76, M.G.M. per 100 cc. plasma.

The treatment given was heliotherapy locally to the glands of the neck without any beneficial results. Potassium iodide, starting with one drop t.i.d., and raising it to 10 trops t.i.d. X-ray therapy, without any improvement. Early in 1937, the glands of right side of neck were removed, and a series of intravenous neo-salvarsan were given. In March, 1937, all of the glands both sides of neck were removed. A small fluctuating area was found on the left anterior chest wall, which was incised, and an abscess cavity entered which was 2 cm. in diameter, and extended down to the muscle of the chest wall between the ribs. The specimen smear was negative, but culture on Sabouraud's media showed torula infection. An area of bone destruction without symptoms was found on the ninth rib, right, about two inches from the end of the rib (April 8, 1937), causing a 9 cm. point of destruction. This area was excised.

X-rays of other bones of body showed no bone involvement. Repeated x-rays (the first of which showed the right leaf of the diaphragm to be obscured by pleural involvement) showed a rather uniform increase in density in the right costophrenic angle (pleural thickening) and possible fluid (five attempts at aspiration were negative). The pleural involvement appears to extend into the interlobar fissure. The midportion of the right lung showed a mottled increase in density of the lung tissue. In the apex there appeared a slight clearing of right lower area.

On August 10, 1937, receiving heliotherapy of region of excised neck glands, the patient improved. He was afebrile and ambulant. He received treatment with sulfanilamide, and was also given two 5-hour treatments of hypertherm.

On March 26, 1938, this patient shows healing of cervical region and area of rib resection. His fever therapy was completed. A tuberculin test was found to be positive. X-ray of chest shows findings unchanged. He was fever free, improved and discharged.

The clinical diagnosis, April 1, 1938, was chronic pneumonitis, right lower lobe due to torula histolytica and secondary involvement of cervical glands, chest wall and ninth rib right.

Resume: These mycotic (torula histolytica) lesions, although chronic, undergo resolution and cure in the majority of cases. The signs and symptoms of this disease resemble tuberculosis. There were no indications of a cerebrospinal involvement. Torula organisms were found in the tissues.

CASE 7.—A. G. H., a white male, aged 59, a salesman, was admitted to the hospital on November 16, 1934. Died October 9, 1935.

The past history showed that he had had measles, mumps and pertussis as a child. Had pneumonia, left upper lobe, in December, 1933. Denied luetic infection. Gonorrhea in 1893. Family history irrelevant, smoked excessively, and had had one year as a soldier in the Philippine Islands (1899-1900).

The patient gave a history of an acute sciatic neuritis in 1899. He had a recurrence of the same condition in 1915. In December, 1933, he had a sudden attack, severe knife-like pain in the left chest and an afternoon temperature of 105 degrees. The next day he had a severe cough, expectorated bright blood and some sharp pain in the left chest. The hemoptysis continued for ten days. He felt so badly, complaining of irregular fever, weakness, cough, with purulent sputum and pain in his lower back, that he had to remain in bed. In February, he took sun baths for a period of six weeks and improved. In April, 1934, he was hospitalized and remained until August, 1934; his body was placed in a cast for six weeks, and diagnosed as arthritis. His temperature continued during this period and physiotherapy aggravated his condition. Hydrotherapy improved him so that he was able to walk about. Soon his condition became worse again, he lost weight, had night sweats, a temperature varying from 100 to 101 degrees; the cough was distressing and the back pain became worse.

On admission he was undernourished, poorly developed, looking chronically ill. Weight 128 (normal 160 pounds). Moderate peripheral arteriorsclerosis. He had pain in the lumbar region, extending down over the sacrum, occasionally radiating to the knees and lateral surfaces of both thighs; fever, prolonged; a chronic persistent cough; night sweats; pain in lower right chest; tenderness in left lower abdominal quadrant; blood pressure 106/70; heart normal in size, position and sounds. Chest showed fine rales over both apices.

On June 5, 1935, due to his temperature, increasing leucocytosis, slight rigidity of the upper quadrant, and tenderness on deep

pressure over the liver, either a liver abscess or empyema was suspected, and 30 cc. of fluid was aspirated from the right chest. Malignancy was strongly suggested, possible hypernephroma. He continued to retrogress. He was explored for a liver abscess on June 17, 1935. No abscess was found, but the liver was enlarged. The cough continued, became severe, the temperature ranged from 98 to 101 degrees. X-ray showed an increase in the fluid at the right base, and an increase in extent and size of the stippling previously reported in both lungs. On July 6, 1935, 100 cc. of thick cream-colored pus was aspirated from the right chest. This empyema was frequently aspirated and irrigated with sodium chloride solution and 50 cc. of azochloramid in triacetin instilled. To this was later added basic fuchsin for five days. Empyema was soon cured.

On August 12, 1935, he was given gradual increasing doses of potassium iodide, believing his condition to be one of pulmonary fungus infection, although no such diagnosis had been confirmed by the laboratory until the following week when the fungus of torula histolytica was found. Patient improved rapidly under this treatment. He was allowed to walk a little.

About October 1, 1935, he developed a small area of ecchymosis on the dorsum of his right hand with some edema. Several days later there appeared a marked odema of the scrotum, hand, feet, ankles and face. From this time on his course retrogressed. He was given blood transfusion, and had received approximately 100 cc. of whole blood, by the direct method, when he remarked that his heart was full. He had a slight clonic spasm, became pasty white, stopped breathing, and no heartbeats were noted. By artificial respiration and adrenaline he regained consciousness and complained of being dizzy and nauseated.

For several days his appetite failed and he had only soups and hot toddys. He was given several intravenous infusions of 250 cc. to 500 cc. of 10 per cent glucose, in an attempt to keep up his nourishment, but not to overload the already damaged heart and kidneys. He expired October 9, 1935.

Laboratory Findings: Smears for fungi showed repeatedly positive for yeast cells, and finally torula histolytica. Sputum negative for tubercle bacilli and animal inoculation was also negative. Smears and cultures of right chest showed staphylococcus aureus. Blood cultures (six) were negative. Urine was consistently negative, except for later stages, when he showed traces of albumin, coarse granular casts, and occasional W. B. C. Blood chemistry: sugar 104;

N.P.N. 29, plasma protein 5.7. Gastric analysis, complete absence of free HCL. Blood serology: negative Wassermann and Kahn. Blood counts were within normal range.

Diagnosis: (1) Fungus infection, pulmonary, severe, torula histolytica; (2) pleurisy, chronic, fibrose, involving interlobar pleura, right upper and middle lobes and apex, left, secondary to No. 1; (3) empyema, thoracic, acute, right base, cause undetermined; (4) myocardial degeneration, chronic, toxic. Confirmed by autopsy.

Autopsy: The mediastinal glands are large, dark and matted together. Section shows numerous miliary grayish-white lesions, suggesting tuberculosis. Pleura is densely adhering to the chest wall through the entire pleural cavity. The lungs are solid and nodules are felt within them. Section shows a studding with miliary, nodose, whitish lesions, which feel firm and tend to be confluent throughout. The pulmonary tissue between the miliary lesions is pneumonic and atelectatic.

The Anatomical Diagnosis: (1) Lung, torulosis of, with multiple abscess formation due to torula histolytica (laboratory proven); (2) nephritis, acute, due to torula histolytica, with multiple abscess formation; (3) spondylitis, acute, fourth lumbar vertebra (torula histolytica); (4) pleuritis, right, secondary to No. 1; (5) empyema, right base, secondary to No. 1.

CASE 8.—P. R. G., a married white American male, aged 37 years, was admitted to the hospital in February, 1933. He had had the usual childhood diseases, influenza in 1918, a tonsillectomy in 1920, and an appendectomy in 1929. He denied any form of venereal disease. The family history was irrelevant.

On admission he was ambulant, febrile, and not apparently seriously ill. His general nutrition was good; the chest showed diminished resonance over the left upper, with parenchymal rales; rales were heard also over the right midchest, anteriorly, and at angle of the right scapula, posteriorly; the blood pressure was 100 mm. Hg. systolic and 75 diastolic; his weight was 130 pounds. An x-ray of the chest showed nodose involvement, most marked in the left upper, the right mid-lung, and the right base. The lesions in the right lung had a radial distribution, not typical of tuberculosis. Repeated sputum examinations, including animal inoculation, were negative for acidfast bacilli. Yeast buds were found in direct smears, and in cultures of the sputum, and were also found in smear and culture of material from bronchoscopic aspiration. Bronchoscopy

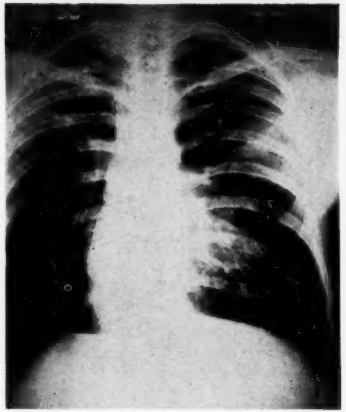

Fig. 22, Case 8. Closely packed small discrete lesions, both lungs.

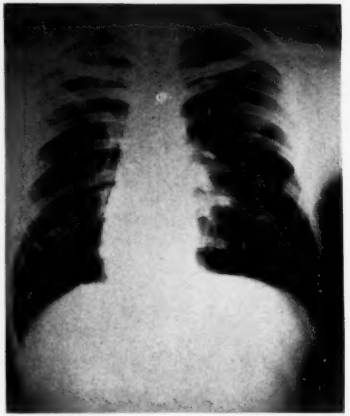

Fig. 23, Case 8. Right Lung.—There is an area of involvement in the outer part of the 2nd and 3rd interspaces. Left Lung.—Above the 3rd rib anteriorly, lesions are small and discrete. A moderate amount of perifocal reaction is present. No cavitation demonstrated.

Fig. 24, Case 8. Radiating outward from the hilum on the left is a density which fades to the periphery. The left apex is homogeneously apaque. The right hilum shadow is also accentuated.

showed moderate congestion of the bronchial mucosa. No free secretion was noted. This yeast was identified as torula histolytica (by Dr. Henrici). It showed on culture a spherical budding yeast of medium size. It did not form spores on plaster blocks, carrot agar or Gorodkova's agar. No sugars were fermented. It was highly virulent for white mice, killing in two or three days, with a generalized peritonitis. The blood Wassermann and Kahn tests were negative. The spinal fluid was negative, and smear and culture showed no fungi. His general condition improved and he left the hospital April 30, 1934. He returned July 13, 1935, because of retrogression. Physical examination was negative (weight 119) except for chest which showed evidence of pathology, bilateral. During this admission he gained in weight to 132 pounds, and was still in fair condition when discharged again in October, 1935.

He returned July 8, 1936, complaining of laryngeal trouble, soreness, hoarseness, and pain on swallowing. The larynx showed a large cauliflower proliferating mass on right false vocal cord, with areas of ulceration. The condition did not resemble a tuberculosis ulceration. The physical examination of chest had changed little. There was a more emaciated appearance, the weight was at this time 122 pounds. The throat became worse, and he retrogressed, showing a wasting of muscles, with partial paralysis of left arm, probably toxic; later, a mental confusion and toxic psychosis developed. For the following month the dyspnea increased. Temperature rose from normal to 100.4 degrees. Pulse became irregular throughout and higher, from 84 to 126. All sputum examinations were negative for acidfast bacilli. Patient finally died in coma.

Laboratory Findings: Repeated x-rays of chest show the progressive fungus type of infection. No definite cavitation is seen. Urinalysis negative. Stool examination negative. Sputum examination (57) tests all negative for acidfast bacilli. Animal inoculations negative for tuberculosis. Yeast buds were found in sputum, and as a result of bronchoscopic examination, as torula histolytica. Wassermann and Kahn negative. Spinal examination negative.

Clinical Diagnosis: (1) Mycosis, pulmonary, chronic (torula histolytica) involving all lobes both lungs; (2) laryngitis, chronic, ulcerative (torula histolytica); (3) tuberculosis, pulmonary, chronic, active, all lobes both lungs.

Note by Pathologist: Acidfast bacilli are demonstrated in smears from both lungs and the prostate. None are demonstrated in the

laryngeal ulceration, but material from the lesion yielded a pure culture of monilia.

Anatomical Findings: (1) Pulmonary mycosis, chronic (torula histolytica) all lobes both lungs; (2) tuberculosis, pulmonary, chronic, active, all lobes both lungs, with cavitation (?); (3) laryngitis, ulcerative, mycotic, severe; (4) tuberculosis, prostate.

Lungs (autopsy): The lungs are voluminous, the right weighing 810 grams and the left 1045 grams. They are much firmer than normal and are very heavy. There is marked emphyema in all the borders. Small nodules are felt throughout the lungs. The pleura is somewhat thickened and roughened where adhesions were separated. All the interlobar fissures are partially obliterated by firm fibrous adhesions. Left lung: The sectioned surface presents a very striking appearance. All lobes are uniformly seeded with innumerable closely placed, small, rounded, grayish-yellow nodules averaging 1 mm. in diameter, but even the firmest areas float. In the apex are numerous small, very thin-walled cavities. Sectioned surface of right lung presents a similar appearance, except the lower portion of the lower lobe, where there is relatively little of the nodular seeding. An occasional caseous or calcified nodule is found, and there is found one thin-walled cavity about 1 mm. in diameter in the midportion of the upper lobe.

Larynx (autopsy): In the larynx there is extensive ulceration which has resulted in the erosion of a large portion of the posterior half of the right cord. The process is very severe on the inferior aspect of the epiglottis, and the entire tip of the epiglottis is absent. The tracheobronchial lymph nodes are very large, grayish in color, soft, and on section appear inflammatory with very little anthracosis. No caseous or calcified nodes are found.

Prostate (autopsy): The prostate is enlarged, and on section, in the left lobe there is found a large, irregularly shaped, poorly circumscribed area of caseation.

Resume: This case previously reported as one of mycosis, showing all evidence of the disease, and a mycotic infection of larynx is evident clinically and by autopsy. This was a case of primary mycosis of the lung, which by the weakness incurred through this infection, the tubercle bacilli found an easy means of rapid proliferation and the death of the patient.

CASE 9.—F. R., a single white American truck driver, aged 28 years, whose family history was negative, stated that he drank some-

times to excess. He was doubtful as to childhood diseases, but stated that he had a severe case of typhoid fever in 1915, and an injury to the left ankle in 1931. He denied all forms of venereal infection.

Present Illness: Patient enlisted in army June, 1920. In November, 1921, he noted that he began to tire easily, had a loss of appetite, pains in chest and moderate cough in the afternoon, fever and night sweats; he lost more than 20 pounds in weight. This condition was not severe enough for him to be admitted to a hospital until February, 1922, when he was admitted and where he stayed until he was transferred to this hospital, arriving October 26, 1922.

On admission, he stated that his condition improved while in the other hospital, he had gained some weight, his temperature had improved, and the present complaints now were headache, moderate cough with expelling of about one-half cup sputum daily, a temperature of 98.2 degrees, pulse 110 and respiration 20. He was 14 pounds underweight with a blood pressure of 135/80. There was an occasional duplication first sound heard in second interspace bilateral. Diagnosis on admission: (1) Observatory for pulmonary tuberculosis and blastomycosis; (2) pleuritic adhesions both bases. In December, 1922, he developed a spontaneous pneumothorax, right. In January, 1923, the observation for pulmonary tuberculosis was changed to pulmonary blastomycosis involving all lobes both lungs. The pneumothorax was practically absorbed. He was placed on K.I. and x-ray treatments, and on heliotherapy during summer of 1923. In October, 1923, general condition much improved, he had few symptoms, no cough or expectoration, maintained his weight. He had no rales in his chest. He was short winded. In December, 1923, he developed a pain and swelling in the left ankle which soon disappeared. A tonsillectomy was performed in April, 1924. His left foot and ankle would occasionally swell, but otherwise he felt good until November, 1924, when he developed a high fever and pain in both sacroiliac joints and left shoulder, and was then diagnosed as a case of arthritis in these regions. A few days later he developed a laryngitis and pharyngitis. In January, 1925, all symptoms cleared up except a moderate sacroiliac pain. In February, 1925, he had a turbinectomy, which was followed by a streptococcus infection of the pharynx and of the left middle ear, associated with a high fever. All symptoms subsided in one week, except pain and discharge from left ear. In April, 1925, a mastoid operation, left, was performed, which was followed by pains in most of joints and regional muscles. In June the general condition improved, but he had swelling in the left ankle if he

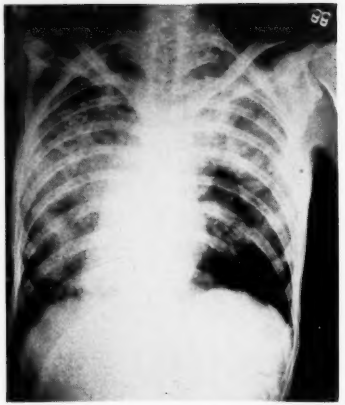

Fig. 25, Case 9. Widespread small miliary lesions throughout both lungs. Small multiple cavitations upper left lung.

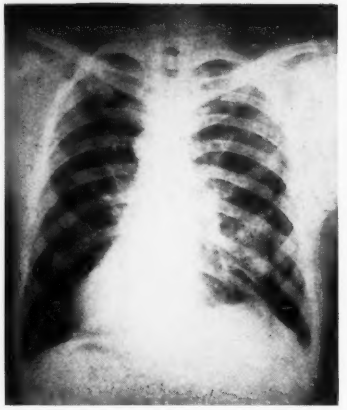

Fig. 26, Case 9. Negative except for mottling suggestive of bronchiectasis, right base.

Fig. 27, Case 9. There is slight thickening of the interlobar pleura between the right upper and middle lobes. A few old lesions, fibrosis, and calcified in mid-lung. Through both lungs there is a fine stippling which suggests fibrosis.

walked. On July, 1925, he was given an intravenous injection typhoid vaccine, which gave a marked reaction, and he refused any further treatment with foreign proteins.

Clinical Course: Observation tuberculosis pulmonary; observation blastomycosis pulmonary (see *Resume*). He had continuous elevation of temperature, 99 to 100 degrees, with normal morning remissions, had a very slight cough with scanty expectoration, and occasional pains in chest, not severe, more marked in left hypochondriac region. His general appearance and nutrition were good. The patient seldom complained, had very few symptoms, and had not lost weight. These symptoms were unaffected by exercise. A physical examination of chest was negative for tuberculosis. An x-ray showed heavy diffuse mottling over entire lungs and reported advanced tuberculosis by roentgenologist. Sputum reports were negative for tuberculosis (17 examinations). It was reported once that sputum contained the budding forms of blastomycetes.

Record showed that the lung condition apparently improved right along, but had stormy course with eye, ear, nose and throat section, and with arthritis.

On November 26, 1925, patient returned from Thanksgiving dinner suffering with much distress and cyanosis, and on November 27th he became critically ill, with symptoms and findings of beginning pneumonia involving right lower lobe. On November 28th, digitalis and oxygen were given. Patient had restless night, very cyanotic, short winded, vomited during the day, and slept very little. On November 29th he developed signs of bronchopneumonia, with dilatation of heart, became pulseless, but heart sounds were loud and snappy over base, muffled at apex, with stethoscope. About 11:00 a. m. heart sounds became irregular and about 1:30 p. m. was very restless, though conscious, he continued to be restless until death suddenly occurred by cardiac failure at 3:05 p. m.

X-ray, October 28, 1922: Right lung, peribronchial infiltration and mottling, moderate, throughout the lungs. Scattered throughout all lobes, apex to base, are moderate number of parenchymal lesions, rather small, and not confluent, appear fibrocaseous and caseofibrous and active. Left lung, moderate peribronchial infiltration and mottling throughout. Scattered through all lobes, similar to the right, are numerous parenchymal lesions, caseofibrous and fibrous, with considerable fibrosis in the upper lobe. Lesions are active, oldest lesions in upper lobe of this lung.

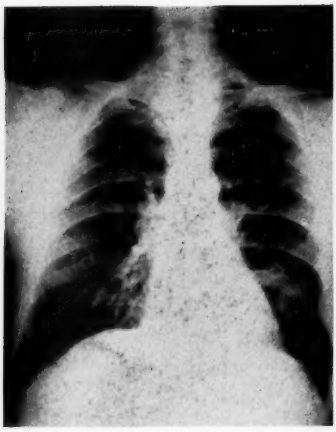

Fig. 28, Case 9. Mottling, peribronchial in type, through the middle portion, which is not characteristic.

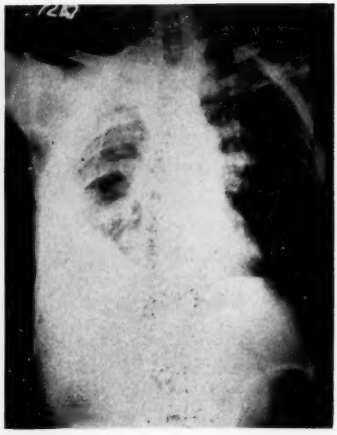

Fig. 29, Case 9. There is miliary type of mottling in both lungs, more on the right. The area at right base suggests a pleuritic shading, perhaps a sequel of pleurisy with effusion.

Impression: Tuberculosis, pulmonary, chronic, all lobes both lungs, active.

X-ray, January 8, 1924: Both lung fields appear clear, with a tendency for the lesions to become better defined and more discrete.

Resume of Laboratory Findings: Urine negative except for occasional trace of albumin and occasional cast. Stool showed yeast cells in several specimens. Sputum showed positive for blastomycosis, May, 1923, January, 1923, March, 1923. Guinea pig inoculation negative for tuberculosis and blastomycosis. All specimens of sputum for tuberculosis negative. Blood count, red count within normal limits, white caries from 9,000 to 24,000, and polys from 78 to 90 per cent. Blood cultures negative. Blood Wassermann in repeated tests all negative.

Lungs (autopsy): Both show easily separated adhesions on all surfaces. A small nodule is felt just under the pleura, and on removal a smear shows tubercle bacilli. Both lungs edematous in consistency, and on section show edema rather than pneumonia. There

are areas of peculiar appearing grayish patches which seem to be a fine network of fibrosis, though not resembling the fibrosis so often seen in chronic tuberculosis. No abscesses are seen, though there are two small cavities about 2 cm. in diameter in the apex of each lung. Rest of the organs show no pathology.

Liver (autopsy): Is large and firm, having the consistency usually found in amyloidosis. On section this opinion is confirmed, with further addition of some fatty infiltration.

Spleen (autopsy): Is somewhat enlarged, and shows a perisplenitis, recent in character, showing a small amount of fibrinous exudate. On section, a small hemorrhage infarct is seen, and surface gives the appearance of moderate amyloidosis.

Kidneys (autopsy): Both kidneys are normal in size and consistency, capsules strip easily and, on section, the markings are distinct and normal in appearance.

Pancreas (autopsy): Apparently negative.

Adrenals (autopsy): Apparently normal.

Diagnosis (autopsy): (1) Blastomycosis (monilia), pulmonary, involving all lobes both lungs; (2) bronchopneumonia; (3) pericarditis, acute, with effusion; (4) cardiac hypertrophy and dilatation; (5) mastoiditis, chronic, suppurative, left, streptococcus. Operation April 16, 1925.

Resume: Blastomycosis is a generic term and is subdivided into (a) monilia, (b) oidiomycosis, (c) coccidiosis, (d) torula, (e) saccharomicosis, usually a contaminant. The pathogenicity is questioned by many authorities.

While the history gives this monilia albicans infection much prominence, nothing is seen which is suggestive of this infection unless the peculiar fibrosis of the lung is a residuum of this infection.

The positive diagnosis of this disease is far from an easy task, requiring time, patience and considerable special technic for its recognition. The organism may lodge in the lymph nodes in the hilus, and necropsy often shows a focus of infection obviously older than the other lesions, and probably the primary focus. In the pulmonary form of this disease, the clinical and pathological pictures are difficult to distinguish from those of tuberculosis.

Fig. 30, Case 7. On palpation the right lung is solid, and nodules are felt within it, but apparently the lung contains no air. Section shows homogeneously studded miliary, nodose, whitish lesions, which feel firm, and tend to be confluent throughout. Innumerable firm lesions the size of the end of the fifth finger and smaller, Torulosis Histolytica, with multiple abscess formation.

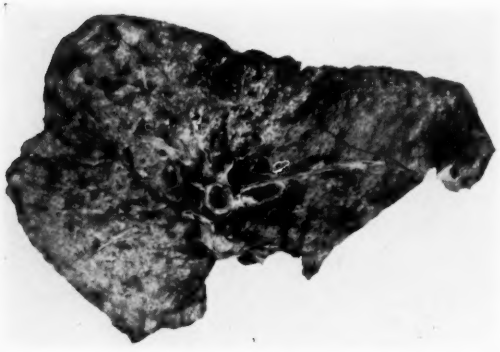

Fig. 31, Case 7. On palpation the right lung is solid, and nodules are felt within it, but apparently the lung contains no air. Section shows homogeneously studded miliary, nodose, whitish lesions, which feel firm, and tend to be confluent throughout. Innumerable firm lesions the size of the end of the fifth finger and smaller, Torulosis Histolytica, with multiple abscess formation.

CASE 10.—L. L. H., male, white, age 33, born in Arkansas, was admitted to this hospital on February 25, 1937, with a diagnosis of (1) fracture, simple, severe, multiple, sternum; (2) fracture, simple, severe, complete, transverse processes of first, second, third and fourth lumbar vertebrae, following a head-on collision with another car. On March 17, 1937, he developed a bronchopneumonia, involving entire right lung, caused by pneumococcus Type II, which was cured April 15, 1937; also, pleurisy, acute, suppurative, right, caused by pneumococcus; empyema, severe, right, pleural cavity, due to streptococcus nonhemolytic following bronchopneumonia, onset April 15, 1937; he was cured October 8, 1937.

On March 19th, 23rd, 25th and 29th, monilia albicans was found in the sputum with the notation that it "killed a rabbit in 48 hours." This suggests significance as regards the pulmonary pathology in this case. On April 6, 1937, the fungus was no longer found.

The differential count March 18, 1937: White cell count 22,350, lymphocytes 22, polymorphonuclear neutrophiles 78. March 19, 1937: White cell count 42,700, lymphocytes 6, polymorphonuclear neutrophiles 93, eosinophiles 1; gradually the count subsided to normal. April 16, 1937: White cell count 9,650, lymphocytes 28, polymorphonuclear neutrophiles 70, eosinophiles 2. Blood chemistry May 9, 1937: Sugar 97, urea nitrogen 14, icterns index 4.2, chlorides 495. Blood cultures, no growths. March 31, 1937, culture of sterile urine showed B. proteins on five different occasions. Urinalysis: straw colored, cloudy, varying in acidity or alkalinity with a specific gravity 1010 to 1017; no albumen, no sugar, a few shreds and occasional W. B. C.

This patient evidently recovered from all the conditions for which he was hospitalized and was discharged.

Resume: It is evident in this case, while he was undergoing the severe convalescence from his bronchopneumonia, the fungus found an easy access to his pulmonary condition. The vitality of the patient was sufficient to overcome the disease and he showed no further manifestation of the mycotic infection at the time of his discharge. The patient has had no follow-up since his discharge.

CASE 11.—G. J. B., a white American male, age 22, was admitted to the hospital on March 16, 1933, with a diagnosis of tuberculosis.

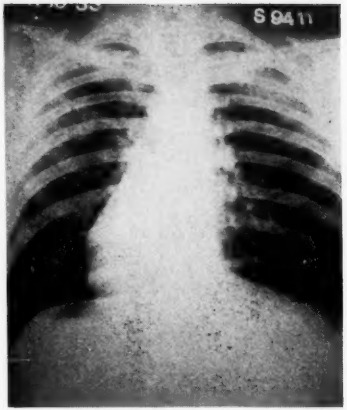

Fig. 32, Case 11. X-ray shows a chronic miliary tuberculosis which suggests a pneumomycosis.

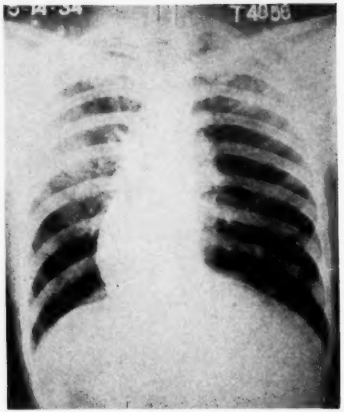

Fig. 33, Case 11. Heavy involvement with small discrete lesions above the 3rd rib right and 4th rib left.

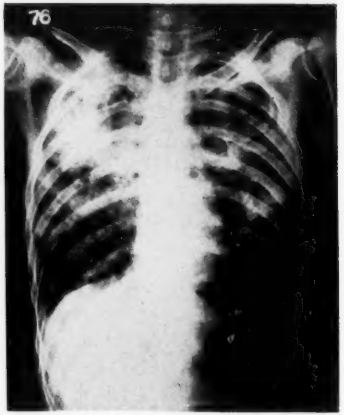

Fig. 34, Case 11. Postmortem. Lesions now confluent in upper right. Widespread discrete lesions lower half of right lung and entire left lung. Extensive excavation upper portion of both lungs.

The previous personal history showed the usual diseases of childhood, St. Vitus dance at age of 9 years, influenza in 1933, pneumonia in April, 1935. He denied history of venereal disease, was a moderate user of alcohol. The family history showed father died at age of 49 with pulmonary tuberculosis.

On admission he gave a previous history of being stiff and sore over all the body. Was diagnosed influenza and lobar pneumonia and was hospitalized in Hawaii for twenty-one days. He was admitted here with the diagnosis of tuberculosis pulmonary. In July 29, 1934, he was diagnosed fungus infection, severe upper lobe right and both lobes left lung (aspergillus fumigatus group). The organism was isolated from the sputum. No tubercle bacilli were found. (Bronchoscopic examination October 17, 1933, negative for fungus culture.) In September, 1934, he developed a burning pain in the rectum, worse during bowel movements, and some blood was expelled. He was diagnosed as rectal tuberculosis. He was operated on, but wound failed to heal.

He left the hospital but was readmitted March 21, 1936; he was ambulatory, febrile, dyspneic and cyanotic. He complained of pain in rectum, throat and abdomen. He had a productive cough, sputum four cups daily. He was hoarse, no hemoptysis, loss of weight (weight 115 pounds, previous weight 165 pounds). Blood pressure 96/106. Severe profuse drenching sweats.

Laboratory Findings: Sputum positive twice for tubercle bacilli, 30 negative for tubercle bacilli. One specimen of sputum was reported on from laboratory in Washington, D. C.: "Specimen identified as member of aspergillus fumigatus group," March 5, 1934. Urine negative. Wassermann and Kahn negative. Blood count, R. B. C. 5,000,000, W. B. C. 13,000, hemoglobin 85 per cent, polymorphonuclear neutrophiles 78.

X-ray Examination, May 18, 1933: Right lung, above the level of the third rib anteriorly, is seen an area of fine nodose fibrous involvement showing perifocal reaction. Left lung, above the level of fifth rib anteriorly, is seen a similar fine nodose fibrous stippling showing perifocal reaction.

Clinical Diagnosis: (1) Fungus infection, severe, upper lobe, right and both lobes left (aspergillus fumigatus group); (2) tuberculosis, pulmonary, chronic active, all lobes both lungs; (3) tuberculosis of larynx and ano-rectal ulcer, tubercules.

Autopsy: The right lung was dotted with nodose subpleural lesions, there is a large irregular, moderately thick-walled cavity in right apex, which communicates with a bronchus and another cavity 3 x 4 cm. that does not communicate with a bronchus and is filled with white, granular, purulent material. The upper lobe is solidly filled with fibrous and confluent nodose lesions, all caseated, but not liquified. The left lung presents an almost similar appearance. Median section shows a large cavity under the apex and occupying about half of the lobe. The upper lobe is solidly fibrous and caseous, with small cavities. No cavities in lower lobe.

Microscopic examination showed caseated areas containing giant cells toward the periphery and surrounding are pneumonic consolidation. The liver, spleen, kidneys and adrenals showed tubercles.

Resume: In this country, aspergillus infection does not seem to be of the pathogenic type and, as in this case, it is always a question of considerable discussion as to whether or not the fungus is purely a contaminate or a secondary invader to tubercle bacilli. It is believed safe to assume that the fungus infection existed prior to the tuberculous infection, whereby the resistance weakened the patient sufficiently so that he became a ready host for tuberculosis.

PERSONAL COMMUNICATION

Thus far we have not heard of any coccidioides infections in your region and shall be very much interested in hearing the results of your tests. If they should be positive, one should be cautious in drawing conclusions, however, and should make certain that the patient has not lived previously at some time in the San Joaquin Valley or some other region where the infection is common, as sensitivity, and therefore positive reactions, persists for long periods after it is once acquired. Furthermore, it should be remembered that erythema nodosum is not exclusively due to coccidioides infection. We have found several people in the San Joaquin Valley with erythema nodosum who were negative for coccidioides infection and proved positive for tuberculosis infection, as well as at least one from whom the coccidioides spherules and acidfast bacilli were both recovered from the stomach washings and both coccidioidin tests and tuberculin tests were positive.

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XXIII

USABLE HEARING*

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St. Louis

Total loss of hearing either bilateral or unilateral, occurs with relative infrequency among the clinical patients which the writer has been privileged to examine. Often those who believe themselves to be totally deaf will hear one or more tones from the audiometer at great intensities. In cases of unilateral deafness, if the good ear be properly masked, the deafened ear may often be shown to have considerable residual hearing in the audiometer test even though the patient be unaware of it. Some may even be able to hear and understand loud conversation; others may hear and understand only words which are shouted quite close to the ear; while still others with less residual hearing may hear the sound of the shouted voice but will be unable to undertand the test words.

Similar reports come from the surveys of the residual hearing of children in schools for the deaf. Hughson, Ciocco and Palmer[1] report that 95 per cent of the children in the Pennsylvania School for the Deaf "respond to at least one tone in one ear by air conduction."

The usefulness of residual hearing of this character depends largely upon the definition of the term "useful." All will agree that the deafened person who is able to hear loud conversation, even though it be at close range, has hearing which is definitely usable. One who can hear and understand words which are shouted loudly close to his ear may perhaps also be considered to have useful hearing. The patient who can hear only the sound of the voice when words are shouted quite close to his ear is in a somewhat different class. It appears improbable that any device could be constructed which would amplify voice sounds so that they would be intelligible to those in the last group without producing additional hearing loss through the traumatic effects of the excessive stimulation. It is true that the determination of hearing losses falling into the last category

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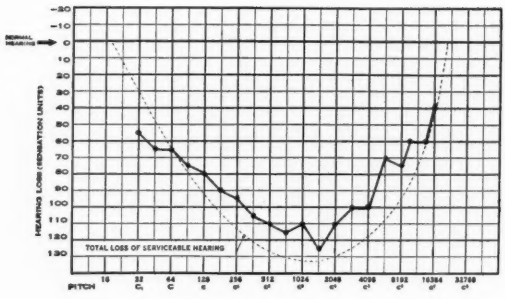

Fig. 1. Audiogram of the Western Electric Co. showing the curve of "Total Loss of Serviceable Hearing" and, in the broken line, the maximum intensity readings for the Western Electric 1A audiometer used in collecting the data of this study.

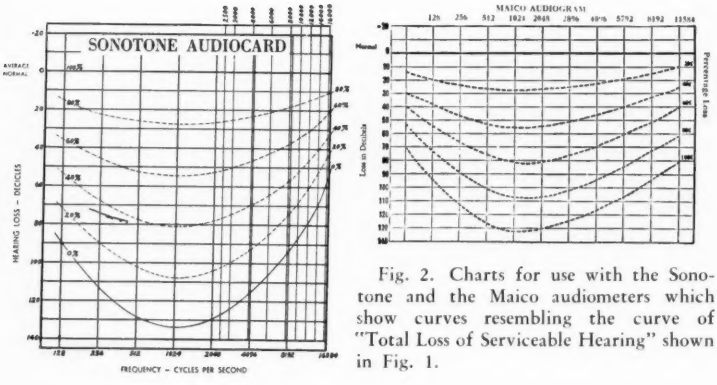

Fig. 2. Charts for use with the Sonotone and the Maico audiometers which show curves resembling the curve of "Total Loss of Serviceable Hearing" shown in Fig. 1.

may be of extreme value in clinical diagnosis, but it seems improbable that this slight amount of residual hearing could be considered "useful" to the patient in any practical sense of the word.

Fletcher[2] of the Bell Telephone Laboratories in 1923 describes the "Auditory Sensation Area" as an area lying between two curves called "Threshold of Audibility" and "Threshold of Feeling," and stated, "When the intensity of a tone is continually increased, a value is reached where the ear experiences a tickling sensation. Experiments show that the intensity for this sensation is approximately the same for various individuals and the results can be duplicated as accurately as those for the minimum intensity value. It was found that if this same intensity of sound is impressed against the finger, it excites the tactile nerve. In other words, the sensation of feeling for the ear is practically the same as for other parts of the body. When the intensity goes slightly above this feeling point, pain was experienced. Consequently, this intensity for the threshold of feeling was considered to be the maximum intensity that could be used in any practical way for hearing."

Later in 1923, on the blank audiograms supplied under copyright for use with the Western Electric 1A Audiometer, a curve is given which is marked, "Total loss of serviceable hearing" (See Fig. 1). Just how this curve was derived is not fully understood by this writer. From the statement in the preceding paragraph it appears that the values may have been derived from the curve of the "threshold of feeling." Whatever may have been the source of the experimental data from which this curve of "total loss of serviceable hearing" was derived, its value in clinical work is of a purely hypothetical nature since no audiometers used for clinical tests produce tones at the great intensities indicated by the curve. Figure 1 shows also the maximum output of the 1A Western Electric Audiometer which was used in securing the data of this study. It will be noted that the maximum output for the tone 1024 d.v. is more than 20 db. less than the output necessary to indicate "complete loss of serviceable hearing."

This curve of complete loss of serviceable hearing has apparently been quite widely accepted by the manufacturers of other audiometers and is used by some for computing the percentage of residual hearing as well as the percentage of loss. Figure 2 shows the charts supplied by the manufacturers of the Sonotone and the Maico audiometers. On both are curves almost identical with the curve of "total loss of serviceable hearing." On the Sonotone Audio-

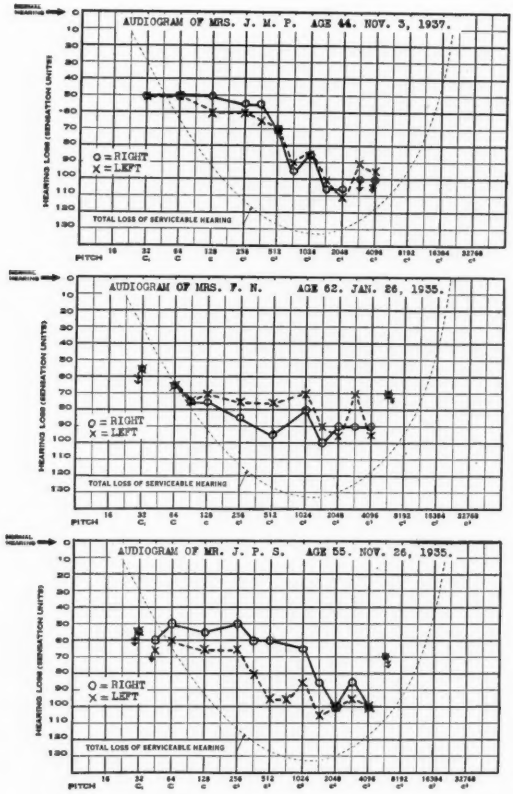
AUDIOGRAM OF MRS. J. M. P. AGE 44. NOV. 3, 1937.
O = RIGHT
X = LEFT
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF MRS. F. N. AGE 62. JAN. 26, 1935.
O = RIGHT
X = LEFT
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF MR. J. P. S. AGE 55. NOV. 26, 1935.
O = RIGHT
X = LEFT
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 3. Examples of the audiograms of patients who were able to hear and understand test words shouted close to the ear. (Small arrows are inserted to indicate that the stimulus tones of the audiometer were not heard at the maximum output.)

card this curve is marked "0%" while the average normal is marked "100%." On the Maico Audiogram a similar curve is marked "100%" with "percentage loss" indicated in a vertical line at the right.

These discrepancies or lack of uniformity lead to the belief that a more practical and perhaps more useful correlation might be desirable for clinical work. Recently Steinberg and Gardner,[3] also of

the Bell Telephone Laboratories, published a curve and state, "Total deafness for speech occurs for an average hearing loss of 85 db." The data for this study was obtained "From laboratory tests upon deafened observers and loss in the capacity for understanding speech in everyday life as revealed by deafness reports in the recently published survey of hearing in the population by the United States Public Health Service." This curve, although falling at much less intensity than the curve of "total loss of serviceable hearing" appears to have a distinct useful value in that it correlates "total deafness for speech" with the audiometric tests. Since the term "serviceable" has already been adopted, another name might be suggested for the curve indicating the total loss of ability to hear and understand speech sounds. The data presented here also tends to verify the practical value of a curve which will locate the intensity limits in audiometer tests beyond which residual hearing is of no value in interpreting speech.

It has been the custom of the writer, in testing patients with profound deafness, to record the results of the audiometer test and to check this result by attempting to determine how well the patient can hear the spoken voice. Many will hear and understand words shouted loudly close to the ear. Others with greater loss can hear the shouted voice but will be unable to hear enough of the voice sounds to understand the test words. The records used in this study were secured from tests of patients whose deafness had been acquired after they had learned to understand speech. Records of congenitally deaf children and of those who had acquired deafness before the habits of speech had been formed have not been included.

In the course of a number of years a considerable number of records of this type have been accumulated. Figure 3 shows the audiograms of three cases who were able to hear and repeat words shouted loudly close to the ear. Figure 4 shows the records of three cases who could hear the voice but could not repeat the shouted test words. Records of 19 ears similar to those shown in Fig. 3, and 32 similar to those shown in Fig. 4 have been secured and form the basis for this study.

The recent action of the Council of Physical Therapy in adopting minimum standards for acceptable audiometers[1] necessitates certain explanations concerning the 1A Western Electric audiometer which was used in these tests. This instrument has been in use in the Otological Clinic of Washington University for over ten years. The intensity values and accuracy of this instrument have not been

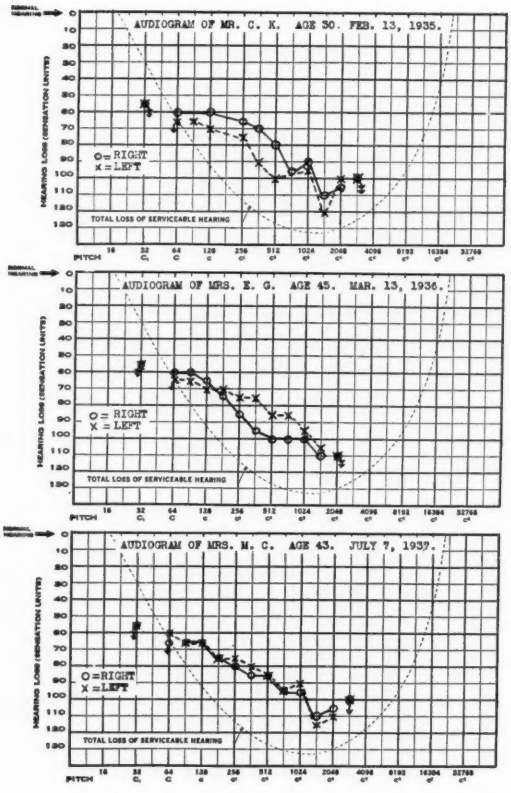
AUDIOGRAM OF MR. C. K. AGE 30. FEB. 13, 1935.
O=RIGHT
X=LEFT
TOTAL LOSS OF SERVICEABLE HEARING
AUDIOGRAM OF MRS. E. G. AGE 45. MAR. 13, 1936.
O = RIGHT
X = LEFT
TOTAL LOSS OF SERVICEABLE HEARING
AUDIOGRAM OF MRS. M. C. AGE 43. JULY 7, 1939.
O=RIGHT
X=LEFT
TOTAL LOSS OF SERVICEABLE HEARING

Fig. 4. Examples of the audiograms of patients who could hear the sound of the voice when words were shouted close to the ear but were not able to understand and repeat the test words.

checked by the Council of Physical Therapy. However, repeated tests on many patients during this time lead to the belief that its accuracy has not altered appreciably.

The records of the first group (19 ears) have been arranged in the form of a scattergram and are shown in Fig. 5. The scattering toward the maximum intensities appears to be quite definitely limited. The heavy broken line has been drawn empirically to indicate the limit of the scattering for the different tones. It may be

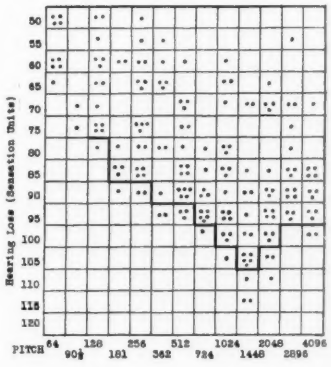

Fig. 5. Scattergram secured from the records of patients who were able to hear and repeat test words which were shouted close to the ear (19 ears).

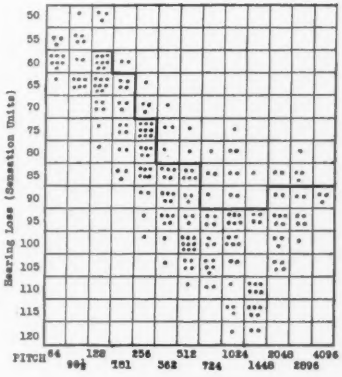

Fig. 6. Scattergram secured from the records of patients who were unable to repeat test words shouted close to the ear (32 ears).

said that all who hear the audiometer tones at intensity values less than indicated by this broken line can hear and understand words shouted loudly close to the ear and therefore have "usable" hearing.

This scattergram and that shown in Fig. 6 give no readings for tones below 64 d.v. or for tones above 4906 d.v. Very few of the patients whose records are included could hear 64 d.v. This is because the maximum output of the audiometer at 64 d.v. is but 65 db. (see Fig. 1). Likewise, the tones above 4906 d.v. were not heard at their maximum intensity. Tones below 32 d.v. and above 16384 d.v. are not produced. The lowest tone specified in the minimum requirements set up by the Council of Physical Therapy is 128 d.v.

Figure 6 shows a similar scattergram secured from records of the 32 ears included in the second group, those who were unable to repeat test words which were shouted close to the ear. As in Fig. 5, it will be seen that the scattering is limited quite sharply. The heavy broken line is again drawn empirically to indicate the minimum limit of the scattering. It may be said all who require intensities greater than are shown by this broken line can hear shouted voice but do not have sufficient residual hearing to understand words shouted

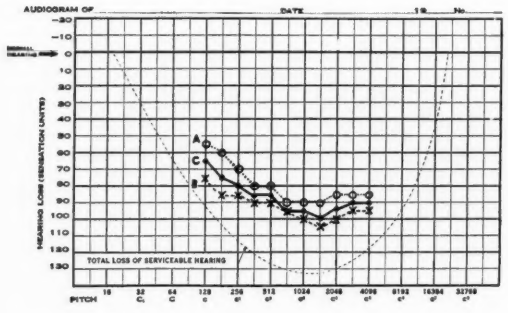

Fig. 7. The curve *A* is drawn from the heavy broken line shown in Fig. 6, *B* is from Fig. 5, *C* is drawn midway (to the nearest 5 db. point) between *A* and *B*.

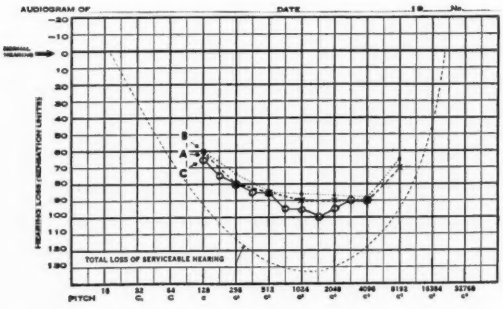

Fig. 8. The curve *A* shows the maximum intensities necessary to meet the requirements for acceptable audiometers established by the Council of Physical Therapy. *B* is the curve established by Steinberg and Gardner. *C* is taken from Fig. 7 of this study.

loudly close to the ear and in consequence do not have "usable" hearing.

Comparison of the two scattergrams show that there is a certain amount of overlapping between the records in two groups. This is illustrated in Fig. 7. The curve *B* is taken from the heavy broken line of Fig. 5 and the curve *A* from the broken line of Fig. 6. The area between the curves *A* and *B* lies in the field of both groups.

This area is 25 db. wide at 181 d.v. and 5 db. wide at 724 d.v. The curve *C* is drawn, again empirically, to the nearest 5 db. point (5 db. is the smallest division indicated on commercial audiometers) to represent a compromise between the records of the two groups. If one remembers that the curves *A* and *B* are drawn empirically and that clinical tests with modern audiometers can be reproduced in repeated tests within an accuracy of from 5 to 7½ db., it appears that the curve *C* is a just compromise. On the basis of this evidence it may be said that if an audiogram shows losses above the curve *C*, i. e., at intensities less than the indicated values, the patient will be able to hear loud shouted words and will have "usable" hearing within this meaning of the word. If the audiogram shows losses which lie below this curve, the hearing will not be "usable."

Figure 8 shows a comparison between curve *C* (of Fig. 7) and the curve *B* secured by Steinberg and Gardner mentioned above. If one considers the varied nature of the source of the data from which the two curves were derived, the close approximation of the two is quite striking. The curve *A*, indicating the maximum intensities for acceptable audiometers specified by the Council of Physical Therapy, has been included for purposes of comparison. It can be seen that the audiometers which do not produce tones louder than the minimum requirements of the Council can scarcely be expected to differentiate exactly between "usable" and "not usable" hearing as defined in this study.

SUMMARY

Evidence is presented for correlation between audiometric tests and tests with shouted voice in cases of profound deafness. On the basis of this correlation a curve is proposed which furnishes, in audiometric tests, a dividing line between "usable" hearing and that which is not "usable."

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XXIV

ESTIMATION OF IMPROVEMENT IN HEARING FOLLOWING THERAPY OF DEAFNESS*

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For many years all therapeutic efforts directed towards the prevention and alleviation of deafness have represented a relatively remote approach to the problem. The chief concern has been the control of infection in the upper respiratory tract and in the ears, its prompt recognition when present, and the immediate adoption of appropriate therapeutic measures. Strangely enough, the hereditary factor was given great importance long before general constitutional disorders were considered as contributory to impaired hearing, regardless of the acute infectious episodes so frequently developed in the establishment of a well taken past history. The pendulum has swung far back in the past ten years or at least swung far the other way. We now know that true hereditary deafness or congenital deafness, in the sense that the parents contributed a mutual defect to the offspring, is probably a rare condition. In the natural course of prenatal development, certain biologic defects occasionally manifest themselves in the normal development of the organ of hearing. These biologic defects render this particular anatomic structure peculiarly susceptible to the trauma of infection or constitutional disease of any character. The breakdown may occur in utero, it may be a neonatal development, or may manifest itself at any time in the first three years of life. Its incidence thereafter is progressively and strikingly less.

In a survery of 313 families of seriously deafened children true hereditary deafness has been established in only five per cent. In only sixteen families were both parents deaf. In thirteen instances

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the father alone was deaf, while in eleven only the mother had impaired hearing. True consanguinity occurred but three times in this series and in these families the mother and father were first cousins. The remaining children represent in order, biologic defects manifested either at or directly after birth, spinal meningitis and upper respiratory and otitic infections directly.

Time does not permit particular discussion of the many new factors advanced in the general conception of the etiology of deafness. Journals are littered at the moment with poorly controlled appraisals of the effects of vitamin and endocrine therapy. No fields demand more immediate or special check than these two, nor is there any field which offers more fascinating speculation. There are many others such as allergy, that utterly subversive influence in the whole scheme of human well being; disordered blood chemistry with its ally, imbalance of body fluid regulation and osmotic pressure change.

Crowe's and Baylor's[1] recent declaration that 50 per cent of human adult deafness can be prevented by elimination of adenoid overgrowth in the eustachian orifice and tube is alluring but ingenuous. Gross pathology, like pure morphologic anatomy, can be now only a matter of historical interest. The classical picture of any disease is an admission that the particular process has progressed beyond the point where therapy can be availing as a truly curative measure.

It is therefore completely requisite that the whole problem of deafness, individually and collectively, be approached from the standpoint of early differential diagnosis. As otologists it is not enough after routine examination to affirm the opinion of the patient that he or she is actually deaf, and for their information and satisfaction tell them the utterly meaningless and still widely used percentage of hearing loss which they happen to have sustained.

Each case must be studied with complete thoroughness and the extent and type of hearing loss determined. All contributory factors must be established and appraised, and resultant disability and consequent economic factors analyzed. And finally, and probably of greater importance, the psychologic reaction must be examined in detail.

Following such an analysis with methods to be detailed shortly, the possible benefit of therapy must be considered. No group of seriously handicapped people is so susceptible to suggestion as the hard of hearing. It is common experience that with any form of

treatment these patients can be made to feel that their hearing is definitely improved when in reality no change in acuity has actually taken place. The reaction to this built-up hope is always severe when realization of the persisting impairment finally dawns upon them.

Therefore, in view of the growing enthusiasm for the newer methods of therapy for deafness, it behooves the otologic group to establish for themselves and for their patients standards of measurement whereby improvement in the hearing level can be properly appraised.

The present methods available for adequate examination are familiar to all. The older technic of examination—tuning fork, watch tick and spoken and whispered voice—however skillfully employed can scarcely be considered methods of great precision and certainly cannot serve as a basis for comparison of observations carried out by different individuals. The fork is quite as susceptible to variation as are the newer pieces of electrical apparatus, but these electrical changes are probably more easily adjusted. In addition, the observer's own hearing may well prove a source of error in examination. We are here concerned with exact measurement of differences in the hearing level, not the simple confirmation of an already established hearing loss.

However many arguments may be advanced that threshold audibility for discrete tonal frequencies is not a true estimate of speech hearing, nevertheless these threshold determinations do indicate a change of hearing at these low levels and as such can be used to measure the effect of therapy. The discrete frequency audiometer, when properly operated and controlled, is an instrument of precision far superior to any other for measuring hearing.

Unfortunately, the reliability of audiometers available at the present time leaves something to be desired. Standardization, though now fairly definitely set, is far from a realized fact, and it is still sometimes impossible to compare accurately audiograms made on different machines by different operators.

The selection and operation of audiometers has already been outlined[2] and their desirable features emphasized. The actual selection of the instrument must be left to the preference of the individual otologist after observed performance qualifies it as meeting standard requirements. In this connection should be emphasized the importance of maintaining uniform conditions in which the

audiometer is to be operated. Recent conversation with an otologist illustrates this point. In the particular clinic where he worked two audiometers were available, one in a reasonably quiet room, the other in the ear, nose and throat clinic where the general noise level was a completely unpredictable factor. Audiograms taken on the two instruments while sometimes checking closely, at other times showed marked discrepancies. Though a sound-proof room may not be absolutely essential there is no doubt that its controlled noise level goes far towards contributing to the accuracy of any hearing test. As previously stated,[2] "In spite of the fact that the patients exist in noisy surroundings, there is no advantage in measuring their hearing under such conditions. The otologist should regard the sound-proof chamber for hearing measurement as a valuable adjunct to his office equipment. It can be dispensed with under certain conditions, but is a great aid to proper interpretation of audiograms."

The necessity for proper masking, when indicated, should be mentioned at this point. An appreciable percentage of air conduction audiograms show the poorer ear to have the same general hearing curve as the better ear or show losses for one or more of the frequencies at which the poorer is reduced more than 30 decibels below the better ear. This phenomenon is referred to as shadow curves or shadow points respectively. The existence of this phenomenon causes doubt as to whether the hearing in the poorer ear, as recorded by the audiogram, is real or is an artifact due to the tone being heard in the better ear by transmission from the opposite side. A repetition of the test of the poorer ear in the presence of a masking sound on the better ear will prove definitely whether the poorer ear is hearing or not. In general, differences smaller than 50 decibels are not shadows, but exceptions to this rule occur sufficiently often to make the use of masking advisable for differences greater than 30 decibels (Fig. 1).

When the audiogram indicates a hearing difference between the ears of 30 decibels or more at any frequency, the better ear should be masked. If the remeasurement with masking gives the same result within plus or minus five decibels the poorer ear is actually hearing the sounds. If the hearing loss is shown to be greater with masking or if the hearing disappears, the sounds were being heard in the better ear. Occasionally, audiograms occur in which the hearing curves differ by 30 or more decibels for only part of the tested hearing range. Only the shadow points need be tested with masking.

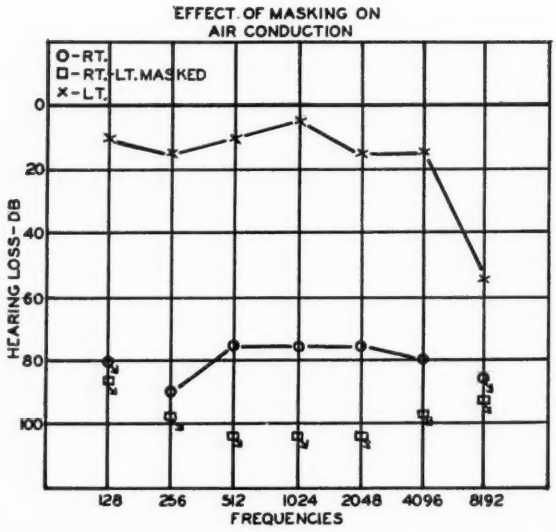

Fig. 1. Effect of masking on pseudo hearing in a case of unilateral total deafness. The five frequencies originally heard in the right ear were shown to be purely a shadow curve when masking at 75 decibels was applied to the relatively normal ear.

Though bone conduction receivers for audiometers are by no means perfected at the present time, certain of the receivers available can be used with very considerable accuracy and satisfaction, and here again the necessity for masking in all bone conduction tests should be emphasized. Under ordinary circumstances it is possible to repeat bone conduction audiograms with little greater variation than that found in the air conduction measurements. Though standardization of bone conduction receivers and the normal threshold level is not established, it is quite possible to determine for an individual bone conduction receiver, fulfilling the major requirements for such an instrument, a level conforming to an approximated normal.

In this connection must be considered the relationship between bone conduction and nerve deafness. It has been shown experimentally that factors other than actual neural damage may seriously impair bone conduction.[3] Quite recently Crowe[1] has confirmed this

observation clinically in his reported series of tests upon young children with impaired bone conduction which was restored by irradiation of the eustachian orifice. The proof of a neural lesion can be only in the confirmation which bone conduction findings give to other types of examinations. This holds except in the obvious neural lesions of old-age deafness or acute toxic conditions.

The so-called loudness balance test in a patient whose ears exhibit a definite difference in threshold level is probably the most important differential diagnostic procedure. Unfortunately, the method is not generally available. In cases of conductive deafness the difference in loudness between the two ears remains the same, regardless of intensity. Where the lesion is of neural origin, at high intensities of stimulation, whatever the original discrepancy in the threshold level between the two ears, the acuity of the poorer ear will approach the better ear. Theoretically and from a physiological standpoint this phenomenon is entirely sound. The recoupment theory, which is based entirely on the loudness theory of Fletcher and Munson,[4, 5] assumes that definite numerical aggregates of active nerve endings must be stimulated in order to produce a given sensation of loudness for a single frequency or band of frequencies. It also postulates that the number of nerve endings stimulated is a definite function of the intensity of the sound stimulus. The hypothesis thus resolves itself to the fact that nerve deafened individuals suffer subnormal hearing because fewer of the nerve endings stimulated by the sound are capable of transmitting their impulses. If a few nerve endings at that portion of the basilar membrane which vibrates to some specific frequency are inactive, obviously the deafened individual will experience no loudness perception when the ear is stimulated with a sound of sufficiently low intensity to be sub-threshold to the deafened observer. Now, if the intensity of sound stimulation is increased considerably above the threshold of the deafened individual, both individuals will experience loudness but the sensation will be in proportion to the nerve endings rendered active by the sound. Furthermore, there will be only a small difference between the number of active endings in the normal and deafened and hence the difference in loudness perception will be small. This is shown more clearly by assuming for purpose of example an arbitrary nerve loss in an individual. Suppose there are 100 inactive nerve endings at some point on the basilar membrane of a given individual. Suppose also that this corresponds to 100 units of loudness lost to this individual over the normal so that there will be no perception whatever until the sound has acquired a loudness of 100 units. The normal

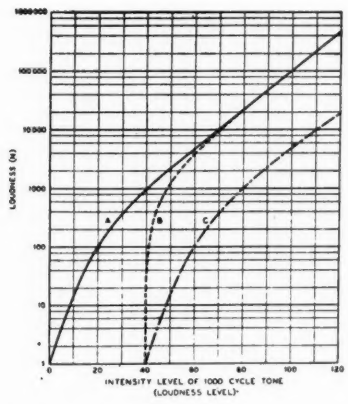

Fig. 2. Loudness versus intensity level of a 100-cycle tone for (A) normal, (B) nerve deafened, and (C) conductive deafened observers. (After Steinberg and Gardner.)

individual, however, will receive a loudness of 100 units at this time. We then increase the normal loudness to 10,000 units, which changes the perception to the deafened individual to 10,000 minus 100 or 9900 units of loudness; 99 per cent of the loudness perceived by the normal. Thus, while the subnormal deafened individual perceived 0 per cent of the loudness received by the normal for loudnesses less than 100 units, he now receives 99 per cent for a loudness of 10,000 units, indicating a recoupment of his loss to a point where he hears as well as normal for all practical purposes. Although the choice of figures in this example is entirely arbitrary and not intended to approach actual conditions, this might well correspond to an individual with an audiometric loss of approximately 20 decibels.

This phenomenon does not exist in cases of conductive deafness where the middle ear attenuates the sound before it enters the inner ear, in which case the attenuation is constant for all intensities, and the inner ear behaves as a normal. Hence the difference in number of loudness units increases with increasing intensity, thus maintaining a constant fraction with respect to the normal. Figure 2 (after Steinberg and Gardner)[6] illustrates the two types of losses, showing the manner in which the nerve deafened loudness approaches the normal when the loss is 1,000 units and the constant parallelism

which exists between the two curves in the conductive deafness where there is no inner ear loudness loss.

Physical measurement of recoupment is accomplished by applying sound to the two ears alternately and requesting the observer to judge equality of loudness. The increment between the sound intensity required to produce loudness parity on the poorer ear over that required on the better ear is observed as a function of increasing intensity applied to a normal ear. If the increment decreases with increasing intensity, nerve deafness is indicated. On the other hand, if there is no change in the increment, conductive deafness is inferred. We have dubbed the apparatus with which this measurement is accomplished an "Omniloudness Audiometer." It consists essentially of two telephone receivers (one for each ear) and two attenuators for reducing the sound to threshold on the two ears. The receivers are held on the ears by means of a double headband and a measurement of threshold made first on the poorer ear with one attenuator (A) and then on the better ear with the second attenuator (B), used in addition to (A), keeping (A) fixed on the threshold value for the poorer ear. (B) is then adjusted until threshold is obtained on the good ear. The reading of (B) thus shows the difference between the two ears directly. The differences for above threshold sounds are measured by first decreasing A by about 10 decibels and then alternating the sound between the two ears by means of an automatic timing device. (The sound is maintained on each ear for a period of approximately one second.) Attenuator B is then adjusted until the observer considers equal loudness to have been attained. Attenuator A is then reduced by another 10 decibels and the balancing procedure repeated, etc. These results when plotted are shown in Figs. 3 and 4.

On one practical point emphasis must again be laid, namely, the necessity of there being a disparity between the thresholds of the two ears of at least 15 decibels. Fowler[7] has stated that this was not necessary, but experience has shown quite definitely that the expected variation in loudness estimation is in the range of plus or minus 10 decibels, which could well make questionable any results obtained from differences less than the level stated above.

When the results of such a loudness balance and bone conduction findings conform to each other, an absolute diagnosis of either conductive or nerve deafness may properly be made. In a relatively small series of cases, loudness balances and audiometric bone conduction tests have been in conformity in 88 per cent of the cases where

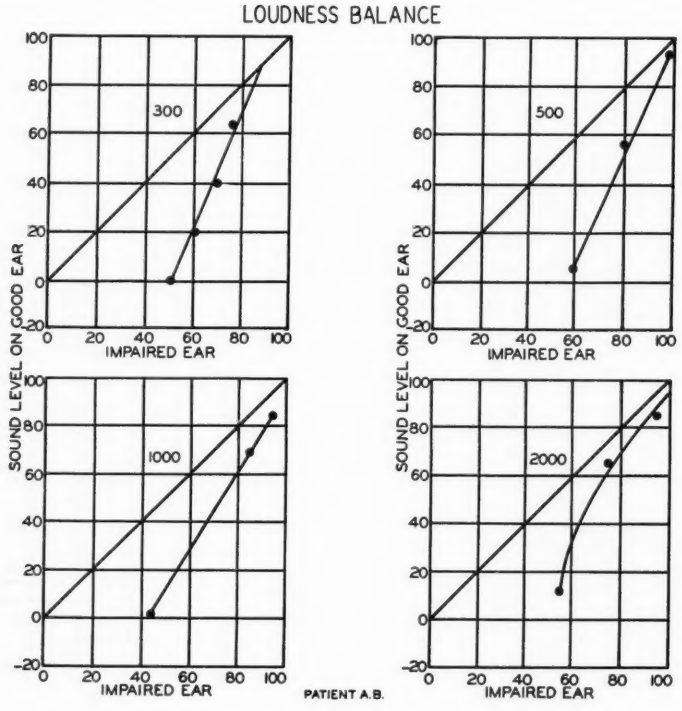

Fig. 3. Loudness balance of variable type. Note that at the higher intensities of the frequency employed the response level of the poorer ear gradually approaches that of the better one, while at maximum intensity the ears respond equally well. This is an instance of nerve deafness.

bone conduction has been reduced and 60 per cent where bone conduction is closer to normal than air conduction. In other words, bone conduction audiograms are reliably accurate in 88 per cent of nerve deafness cases only when the finding is confirmed by a variable loudness balance. When bone conduction approaches normal the accuracy is only 60 per cent.

If a further refinement in the diagnostic technique is desired, fatigue of the ear is a useful procedure. Exposure of an ear, impaired by a conductive lesion, to discrete frequencies of high intensity will not result in fatigue; while if the lesion is neural, fatigue will

result. Any frequency from 512 up to 2048 cycles may be used at an intensity 80 to 90 decibels above threshold. The fatiguing tone is applied to the ear immediately after a standard threshold audiogram has been made. After ten minutes of such stimulation a second audiogram is made and if fatigue develops it will be manifested at the stimulating frequency and those above it in the audiometric range; fatigue never occurs at a lower frequency level. Unfortunately this test is useful only in moderately impaired ears, as most available sound sources have not sufficient intensity range to fatigue a seriously deafened ear.

The final check upon impaired hearing and its relationship to individual frequency loss is the determination of speech intelligibility. The three frequencies concerned, of course, are 512, 1024 and 2048 cycles. However, some frequencies of higher range and less than octaves in extent, are undoubtedly important.

To meet the general objection to threshold audiograms as true measures of speech hearing, an effort has been made to determine the threshold of intelligibility of deafened ears. Such a test made before the initiation of any form of therapy should prove an invaluable check upon the effect of the specific treatment.

An intelligibility test is one method of measuring the intelligence of groups of connected words combined into single sentences. It is quite obvious, that while disconnected words spoken by one person and received by another form a more scientific measure of the vowel and consonant combination heard and understood after passing through any medium of transmission, they can carry no meaning of themselves. It is quite universally acknowledged that deafened persons are interested in hearing aids almost entirely for their efficacy in transmitting intelligible speech. Hence, if the key words of a question or statement can be heard and understood, the meaning of the sentence may be clear and the secondary words which have been missed can be interpolated. This then will constitute the capability of the transmitting medium in so far as the deafened individual is concerned.

The actual mechanics of the intelligibility tests, which were originated by Bell Telephone Laboratories, consists of repeating disconnected sentences to the listener and receiving either a reply (which is not recommended due to the controversial and ambiguous nature of many sentences which may be used) or the sentence repeated by the listener as he understands it. By scoring the number

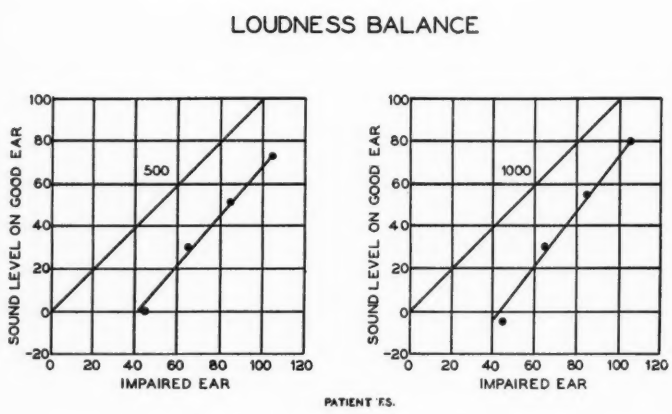

Fig. 4. Loudness balance of constant type. Here the attenuation of loudness lies entirely in the middle ear and is constant regardless of the intensity of the tone used. This is typical of a pure conductive deafness.

heard correctly and the total number repeated the percentage correct may be obtained and the transmission system rated.

The measurement is made in the following manner: An approximate threshold of the patient is obtained for speech with a speaker reading into the microphone of a high quality sound system in which is incorporated a calibrated attenuator. The observer (patient) should be cautioned not to attempt to understand the reading matter but merely to hear it. The sound level should then be set for an intensity approximately 10 decibels above the threshold and about 10 intelligibility sentences spoken. It is possible to estimate from the number repeated correctly by the listener the probable percentage which would be obtained from a list of 50 such sentences, and it is well to strive for approximately 55 per cent heard correctly. If this is reached for the forementioned sound level of 10 decibels above threshold, a complete test of 50 sentences may be carried on. If the percentage is too high or too low the level must be changed accordingly before the complete list, which comprises the test, is used. Several lists should be available and a different list started each time a new intensity is used. Fletcher and Steinberg have published 49 intelligibility lists.[8] It is advantageous to perform two or more tests at different times before therapy is instituted in order

to determine roughly the error and spread of results to be expected from the individual. Once the amplifier attenuator setting which corresponds to the proper intensity has been ascertained, it should be used for all measurements henceforth and a new threshold should not be made. The purpose of the measurement is a determination of changes in ability to understand confluent speech before and after therapy regardless of changes in threshold. Conditions such as voice intensity, distance from microphone, etc., should be maintained as constant as possible for all tests. A volume indicator is a valuable aid in this respect. Theoretically this type of test should tend to prove or disprove the validity of subjective improvement which may not be apparent from audiometric measurement. The intelligibility increases rapidly with increasing intensity for a good transmission system, and if the system alone is to be measured, an intensity greater than 40 decibels above threshold is the only requirement (Fig. 5).

Having arrived at such an audiometric diagnosis, a general constitutional appraisal must, of course, be made, and where defects are found, appropriate measures taken for their correction.

STANDARDS OF IMPROVEMENT

For the past two years papers have been read and published with increasing frequency upon the efficacy of some novel endocrine or vitamin preparation, or upon the benefit derived from some more or less complicated surgical procedure. It is not the purpose here to discuss the relative merits of any specific form of therapy, but rather to establish a standard whereby it can be determined whether the procedure has true merit.

A base line has been established by the various diagnostic measures listed above, the type of deafness determined as conductive, neural or mixed in character. Some type of treatment, assumed to be appropriate, has been instituted and after a reasonable lapse of time an appraisal is reported of the therapeutic benefits. Difficulty of technical methods and the hazards involved, whether medical or surgical are not important, if the promised benefit is great and conversely where the chance of improvement is slight there can be no possibility of risk. No therapy of deafness is warranted where there is anything more than an outside chance of further impairment, nor where, as a result of treatment, ultimate recourse to the use of a hearing aid is jeopardized. Nor should any type of treatment obviate the possibility of benefit by some later more effective procedure.

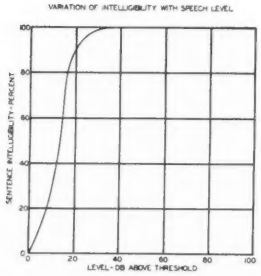

Fig. 5. Variation of intelligibility as a function of sound level. Note the rapid increase of intelligibility with moderate increases in loudness, a maximum being reached at approximately 40 decibels above threshold.

Immediate comparison between pre- and post-therapeutic hearing levels is more a matter of concern to the physician than the patient. In the surgery of deafness this particular phase provides either drama for the observer and patient or a period of often painful waiting until some effect may become manifest. This latter situation is always present in any medical type of treatment. However, from a scientific point of view, such immediate records should be made promptly and frequently. Particularly in all surgical cases there should be as immediate and frequent tests of the hearing level made as is compatible with safety of the final result. Only by such repeated examinations can any true picture of therapy be gained.

Air conduction thresholds are determined with less difficulty than any other type of test. Bone conduction, though of possible interest, has not been affected by treatment with the exception of Crowe's series of cases and in his report the 512 cycles fork is the standard of bone conduction acuity. Intelligibility tests should be repeated as promptly as possible.

We have shown that in the untreated deafened ear an expected variation in response in different tests may average plus or minus 4.2 decibels and show extremes of from minus 14.6 decibels to plus 22.8 decibels. Normal ears exhibit a variation quite comparable but with lesser extremes. Theoretically intelligibility varies directly in proportioan to the change in response at the critical frequency range. This, however, can only be determined by actual tests of

speech intelligibility at fixed intensity levels. Such measurements should be made at stated intervals. They are often burdensome for the patient and always time consuming for the physician but are essential in any careful appraisal.

In view of expected variation, therefore, it is felt that no change in the hearing level of any frequency less than 10 decibels can be considered a matter of the slightest consequence. Attention should here be brought to the fact that fractional changes between 5 and 10 decibels are of no significance unless derived from repeated audiograms, since in all audiometric calibration 5 decibels is the unit of change of intensity.

Quite naturally an ideal improvement in hearing resulting from medical or surgical therapy should be permanent. At the present time it is probably impossible to effect an actual cure of deafness by any means available. If, therefore, a patient submits himself to a long continued course of medical treatment or to the hazard of surgery, there should be reasonable promise that a minimal improvement at least would be relatively permanent.

Unfortunately, few modern methods of treatment have been in use long enough to permit any final judgment in this matter. The human ear is constantly exposed to risk of injury by many factors and therapeutic improvement might easily be lost as a result of any intercurrent infection or development of some constitutional disorder. Careful appraisal of such an accident would be essential in arriving at any accurate estimate of duration of improvement in a given case. In the majority of pathologic conditions which have undergone treatment the "five year cure" has become more or less the standard interval for a satisfactory end result. Everything else being equal it would seem reasonable that the patient should have assurance of maintaining any gain obtained for at least this length of time.

As stated above, the subjective reaction of patients with impaired hearing is an extremely labile one, influenced by any number of factors, chief of which may be the physician's own enthusiasm for his efforts. It is not humanly possible for the otologist to view with complete calm, much less skepticism, a frank improvement in the level of the audiogram or of any individual frequency when it does appear. However, if after a year's time has elapsed when the stimulating novelty of the situation has worn off and the patient still feels that definite improvement is present, the subjective report may be accepted. Probably of more importance is the actual hear-

ing of sounds which were previously quite inaudible. Also in this connection at least a year is the only safe interval between operations on the first and second ear.

Rehabilitation is, of course, the aim for instituting therapy of any kind and should prove, if the effort is successful, to be both social and economic in character. But here any promise we can offer will be entirely dependent upon the original hearing level and the expected improvement of 10 or more decibels resulting from the treatment employed. It is not likely that any level below 30 decibels in the critical frequencies is actually a rehabilitation unless the patient's original occupation made it unnecessary for him to hear speech at normal intensity in quiet surroundings. At present, losses of 50 decibels or more make the achievement of really useful hearing a matter of great uncertainty. Prognosis under such circumstances must be extremely guarded.

It would be a most admirable thing if more otologists would have made a careful psychologic survey of every patient under treatment. This procedure is carried out routinely by Canfield in his clinic and the practice should most certainly be a more general one. A large percentage of deafened individuals have developed, in the course of their handicapped years, certain very definite psychologic reactions often bordering upon profound depression. Improvement in hearing might be expected to relieve this situation, but these patients should be given every possible aid towards reasonable compensation whether or not improvement in hearing does occur.

SUMMARY

In an effort to establish some standard whereby different observers may compare the results of their therapeutic efforts in the management of deafness certain standards of measurement have been suggested. There will, of course, be differences of opinion in regard to the routine merits of individual tests. It is felt, however, that for a completely accurate appraisal, the routine suggested will obviate any criticism of reported results.

The recently awakened enthusiasm for new forms of treatment in deafness, whether medical or surgical, demands that all results be most rigidly analyzed and that there be a common standard of interpretation.

Bone conduction audiograms and loudness balance tests are essential in the selection of cases for certain types of surgery. The

rather general feeling that thresholds of hearing for individual frequencies do not represent the actual hearing level for speech intelligibility is doubtless entirely sound. The routine employment, therefore, of some type of intelligibility test would seem to answer the objection to the standard threshold audiogram.

Of even greater importance is correlation between a minimum standard of improvement and expected variation in repeated audiograms. It should be emphasized also that only by frequently repeated audiograms can any real conception of the effect of therapy be gained, and also that fractional recordings are of value only if they are derived from a considerable series of audiograms.

The arbitrary figure of ten decibels has been chosen as the minimum improvement to be considered of any real consequence and this only if the originally observed level would be actually improved by such a gain in acuity.

Finally the required duration of improvement has been set at five years. No reported group of cases has been under observation for this length of time. Under the circumstances cases maintaining improvement for intervals less than this five-year period should be considered unsuccessful.

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XXV

A SUMMARY OF ROUND WINDOW GRAFT OPERATIONS PERFORMED FOR DEAFNESS*

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A brief review of the history and experimental background of round window grafts in the surgical treatment of deafness might serve to place the procedure on a clearer and more rational basis. The actual function of the round window may still be a matter of some uncertainty but an observed and repeatedly confirmed experimental finding led to the belief that one function of the window and its membrane, at least, was to dampen the intensity of acoustically induced stimuli reaching the perceptive mechanism of the inner ear.

In 1931, following Wever and Bray's report[1] of their studies upon electrical potentials of the cochlea or acoustic nerve resulting from acoustic stimulation of the experimental animal's ear, many workers devoted themselves almost exclusively to the study of hearing with the Wever and Bray phenomenon as a measure of auditory response. The combined effect of these various investigations has been to clarify, in many respects, accepted theories in regard to the physiology of the ear as a whole and of its individual component parts.

One such laboratory was established in the Department of Otolaryngology at the Johns Hopkins Medical School. Here the entire physiology of the ear from the standpoint of air conduction was reviewed over a number of years. Very early in the investigation the function of the round window was studied and, as stated above, definite observations made. Cats under ether anesthesia were used in all experiments. Fixation of the round window membrane by a pledget of cotton, pressed firmly in the niche, invariably produced a remarkable improvement, 10 to 30 decibels, in the hearing acuity of normal ears as measured by changes in electrical potential either from the cochlea or auditory nerve. A very striking effect was

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*From the Otological Research Laboratory, Abington, Pa.

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observed when the whispered voice was used as the sound stimulus to the animal's ear. Before placement of the cotton in the niche no words could be distinguished either in the loud speaker or ear phones of the amplifying system. After fixation of the membrane, however, the whispered words could be recognized with complete clarity and the increased intensity of the high frequencies was particularly apparent.

If the phenomenon developed in the course of an acute physiological experiment, might not the same thing hold true after the round window had been permanently fixed by some surgical procedure? Therefore, living tissue of different kinds, fascia, muscle and periosteum, were used in place of the cotton. The animals were operated upon with strict surgical technic, allowed to recover from the anesthesia and kept for varying lengths of time postoperatively before the ears were tested. In all of these experiments the unoperated ear was used as the control, and after testing both ears were serially sectioned and the material studied histologically. The improved response in chronic experiments was first observed on January 26th, 1932, and in both acute and chronic experiments since that time the effect has been reproduced under a great variety of conditions. It must be remembered that in the animal experiments normal ears exhibited a much greater acuity of hearing following fixation of the round window than did the control or unoperated ear.

These experimental results fell in line with the effect, so often observed clinically, that placement of cotton in the region of the niche in patients with a large perforation of the tympanic membrane often produces a remarkable increase in hearing acuity. The immediate explanation of this response was not readily apparent. Other types of experiments showed that fixation of the round window membrane increased the tendency of the ear to fatigue and increased the extent of damage caused by detonations. This led to the belief that the principal function of this membrane was to serve as a safety valve or safety mechanism to the inner ear structures when the ear was exposed to sounds of high intensity. By fixation, therefore, might not this safety valve effect be eliminated and the intensity of all acoustic stimuli be reflected by an increase in auditory response? Another explanation offered was that, after contraction of the graft and complete fixation had developed, greater reflecting surface in the inner wall of the cochlea was made available. From the electrical standpoint it was known that responses from the oval and round windows were of opposite phase. Fixation of the round window

niche might bring the two responses in phase and thus enhance the total response. And finally the suggestion was made that growth and permanent fixation of the graft might modify intracochlear pressure in some way. It has been shown experimentally that increased intralabyrinthine pressures improve rather than impair acuity of auditory response.[2] However, the inner ear is not a closed fluid system and this hypothesis seems of doubtful importance. At the moment, therefore, there is no valid reason for modifying the original concept.

DEVELOPMENT OF OPERATIVE TECHNIC

In the animal experiments, tissue grafts could be placed in the round window niche through wide surgical exposure and access to the round window was thus made relatively easy. In the human being, however, difficulty of exposure and approach was experienced. Early in the effort to carry out the procedure on human beings it was felt that the condition, impaired hearing, did not warrant major surgery with its attendant hazards. Any technic employed should combine maximum simplicity with a minimum of risk. After various methods, such as forward reflection of the pinna following section through the cartilaginous portion of the external canal, or direct access to the round window through a simple mastoid approach had been tried, a simplified technic of direct approach through the canal and tympanic membrane was developed. Magnification of three to four diameters and adequate illumination are considered essential.

The canal is carefully cleaned of all cerumen and hair the evening before operation. The operative preparation is that used for routine mastoid surgery, and the canal is sterilized simply by instillation of 70 per cent alcohol followed by ether. The canal wall must not be rubbed or traumatized in any way.

Using a standard black speculum, equipped with a handle, to obtain an adequate view of the tympanic membrane, an incision is made along its posterior border as close to the annulus as possible. Bleeding is carefully controlled by suction and application of pledgets of cotton soaked in adrenalin. The flap thus created is turned forward and the promontory and round window niche examined. As has already been pointed out, a direct view of the niche is seldom obtained but the lateral margin can practically always be visualized. The mucous membrane around and, if possible, in the niche, is gently rubbed with dry cotton and the graft, which has been previ-

ously prepared, is packed firmly in position. This degree of firmness is difficult to describe, but there must be no question of rupture of the delicate round window membrane. The middle ear cavity must be free of blood at the end of the operation. Disposition of the drum flap is a matter of little consequence. The flap edge seldom unites with the peripheral margin but rapid regeneration of this margin does take place. Experience has shown that when the membrane is found to be unusually thickened it is better to actually excise the flap to insure rapid closure of the defect. Postoperatively the ear is dressed every third day with rigid sterile technic. Closure of the tympanic membrane usually takes place in two weeks' time.

Though the sequence of events as here described is apparently reversed, the purpose should be clear, namely, that the development of the operative procedure logically followed the experimental investigation of the physiology of hearing. Selection of cases could only become a matter of standard following a reasonably large experience with the operation itself.

SELECTION OF CASES

Every deaf person whose hearing loss makes him an appropriate subject for surgery can obtain immediate and wholly practical help with a properly fitted hearing aid. This point cannot be emphasized too strongly and should give every surgeon pause before he undertakes actual surgery for deafness. But having finally decided that operative measures may be undertaken, the patient must fulfill the following requirements.

Age: Although few patients over fifty have been operated upon by the method under discussion, it is felt quite definitely that this should be the upper age limit, and that all else being equal youth is a distinct asset for favorable results. In only two instances have the patients been under twenty, but it is hoped that application may be extended to this younger age group in cases where the hearing loss has reached a reasonably static level. More women than men present themselves for operation, but certainly sex is not a matter of any real significance.

Type of Deafness: By repeated audiometric tests and the use of special examinations as indicated, prior to operation, it is possible to arrive at a reasonably accurate diagnosis of the specific type of deafness which the patient presents. Air conduction audiograms should be repeated at least three times, allowing a sufficient interval between tests to determine whether extraneous factors influence the

hearing loss either favorably or unfavorably. As has been pointed out, untreated deafened ears show no significantly greater variation in successive audiograms than those with normal hearing. There is, however, a greater possibility of variation in certain types of conductive deafness than in others, and in the conductive group as a whole than in proven preceptive impairment.

A carefully taken history establishes the familial background, the incidence of childhood infections and contagions, the general upper respiratory history of chronic or acute infection, such as tonsils, adenoids, sinus disease, etc. Excessive use of drugs, much less addiction, has been observed in only one case of a large series examined. The age of onset must be accurately dated, if possible, though this is often one of the most obscure points in the entire history. The average patient places the time of onset at the moment when the hearing loss became a distinct handicap and describes this as having happened suddenly. The preliminary gradual loss is seldom recognized.

We must assume that active nose, throat and sinus infections have been eliminated and all possible contributing toxic foci dealt with adequately. In selecting cases for this operation no particular attention has been given the matter of dental malocclusion. Though undoubtedly a contributing factor under some circumstances, it has not been felt that correction in the fourth and fifth decade groups would prove of any benefit.

Infection of the nasopharynx is of great importance, particularly in the adolescent age group. With a nasopharyngoscope small residues of adenoid tissue, either adjacent to or actually within the eustachian orifice, can be identified and a reasonable opinion as to the patency of the tube formulated. It is not felt that patency of the tube is a requisite. Friesner and Druss[3] first pointed out authoritatively the relative futility of eustachian tube inflation and bouginage as a therapeutic measure in deafness. It is further thought that when and if the lumen of the tube is actually obstructed, one possible and active route of infection to the middle ear is definitely eliminated.

Otoscopic examination is regarded as the most revealing single procedure in the entire diagnostic routine. Although fistulization has been reported in cases with a perforated tympanic membrane, it was originally stated and is still maintained that an intact membrane is a prerequisite for the round window graft operation, but

its degree of thickening and retraction need no influence adversely the decision to operate. No tympanic membrane has failed to heal in this series of cases, although in several instances approach to the round window was made through drums exhibiting extremes of retraction and thickening. Fixation of the stapes footplate or immobility of the ossicular chain may be determined by any method decided on by the individual examiner. It is felt that apparent stapes fixation is not necessarily a specific diagnostic factor. Mobility of the tympanic membrane is also a matter of no undue importance.

A most careful physical examination must be made to determine what the general constitutional background of the particular case may be. Aside from the frank old-age arteriosclerotic hypertensives, deafened individuals of all ages seem to fall either in the low normal or true hypotensive group. Though no actual therapy designed to correct the hypotension will affect hearing directly, the relief from fatigue may well modify favorably, to some extent, the inevitable nervous tension and strain of deafness. Experimental studies of body fluid pressures and their effect upon auditory acuity indicate quite clearly that any considerable reduction in pressures tends to impair hearing while increases in pressure, within all physiologic limits, has either no effect or tends to improve acuity.

Vitamin and endocrine deficiencies have yet to be demonstrated satisfactorily as specifically related to impairment of hearing. Compensation for these lacks, though of undoubted general benefit, need not be expected to have any significant effect upon hearing itself.

Allergic states also may affect hearing remotely by the congestion of the upper respiratory mucous membrane and by their effect upon body fluid balance. All allergic manifestations, when possible, should be controlled. Blood chemistry changes should be a matter of record. The cytology and serology of the blood should be studied. One case was operated upon, a young male 18 years of age. The Wassermann was not done until admission to the hospital. Operation was performed before the report of a positive Wassermann was received. The tympanic membrane healed without delay and there were no untoward effects. However, no operation should be undertaken until the Wassermann has been reported negative.

When economically possible, routine x-ray examinations of the sinuses, temporal bones and pituitary region should be made to serve as a matter of record whether disease is demonstrated or not. From stereoscopic lateral and anteroposterior views of the skull as a whole,

information can be gained as to skull contour and density. It is hoped that when sufficient material of this type is available some relationship with bone conduction may become apparent.

When this background has been established it is proper to correlate the findings with the hearing tests which have been carried out during the period of examination. So far as the hearing loss is concerned, it now seems futile to undertake this particular type of surgery on any patient whose general level is below 50 decibels. The greatest improvement obtained in any single instance, and that not a permanent one, has been 30 decibels. This amount of improvement cannot be expected to occur in every case. It would, therefore, serve no possible purpose to operate when the level of impairment was 60 to 70 decibels.

Normal bone conduction in hearing losses of this extent is probably never found, but relatively normal bone conduction with the higher air conduction levels is perfectly possible. However, if the rationale of this operation, as stated above, is reasonably valid, a moderate degree of nerve deafness as demonstrated by bone conduction and loudness balance tests need not prove a contraindication. On the recoupment theory of hearing in the presence of neural damage, moderately impaired bone conduction would not necessarily be a significant factor.

Lately much emphasis has been laid upon the three critical frequencies in the audiometric scale, 512, 1024 and 2048 cycles, as representing the entire frequency range to be influenced favorably by our therapeutic effort. The octaves immediately above and below these two should certainly be added. A large part of speech intelligibility certainly lies in the 2048-4096 cycle interval, while actual loudness is represented in the lower one, 256-512 cycles. This particular octave is always affected more easily and more favorably than the higher one by any surgical means. At any rate it is felt that the two octave range, as stated, is inadequate and not truly representative of complete intelligibility of speech and cannot be the final desideratum of medical or surgical treatment.

In addition to a detailed study of the cochlear division of the nervus acousticus an examination should be made of the vestibular apparatus and from the standpoint of simple routine the caloric test would seem to be sufficient for this purpose. In the absence of actual vestibular symptoms there is no necessity for examining the labyrinth further.

The choice between surgery and a hearing aid must again be discussed. Insistence upon this point depends somewhat upon the age of the patient. The older the individual the less adaptable is he or she to the more unpleasant features of any electrically amplified sounds, particularly when achieved by a carbon microphone. Therefore, if the individual is in the older age group it is more important to become used to a hearing aid promptly than is the case in the younger group. Many specious arguments can be raised against the use of a hearing aid. They should be disregarded. The patients are told by friends that an aid will impair their hearing further and by salesmen that it will improve the actual hearing level. Probably neither is correct. The sole purpose of a hearing aid is to bring a deafened individual into immediate and easy auditory contact with his fellows.

Unfortunately, sufficient time has not elapsed to make a final appraisal of the round window graft operation a matter of enough importance to present at this time. Such a report will be presented at a later date when it is possible to review the series from the standpoint of a four years' experience.

However, the following definite facts are already established on the basis of slightly more than three years' observation and bear out statements made in the original report upon round window graft operations.

In one respect the selection of cases has been further restricted, namely, advanced age has become a more serious bar to operation than was at first supposed to be the case. This fact is also reflected in the duration of the deafness. On the other hand, it is no longer felt that a moderate degree of perceptive deafness, particularly in the higher frequencies, 4096 and 8192 cycles, is a definite contraindication to surgery. Nerve deafness, like a conductive impairment, reaches a static level which may remain unchanged for years.

In the relatively small series of cases operated upon there have been no immediate postoperative complications, such as infection or failure of the drum to heal. Nor have there been remote complications of any sort, such as facial nerve or labyrinthine disturbance, either early or late.

The operation as performed, under avertin anesthesia, is entirely devoid of unpleasant after effects and is quite painless. The hospitalization period is of three days' duration, after which the patients resume their normal activities.

In no instance has the late, six months and more, condition of the tympanic membrane precluded surgery of a different type. No serious scarring has resulted, although in a few instances, after a lapse of almost a year, an area of atrophy has appeared at the site of the incision. The union of the cut edges is entirely secure, as evidenced in one patient who flew several thousand miles at a high altitude the day following observed closure of the opening.

Tinnitus, though modified and changed in certain cases, has never been completely eliminated. In one instance the patient acquired a high-pitched pulsating tinnitus several months after operation.

Bone conduction has been affected neither favorably nor adversely in any instance.

The poorer ear has always been selected for the first operation, and a second operation done only when the level of the two ears had become relatively the same.

Variation in the audiogram of the operated ear occurs with just as great frequency and to approximately the same extent as in the intact ear. It is affected by exactly the same condition.

The striking shadow improvement in the unoperated ear, recently reported by Kopetzky,[4] has not been observed in this series.

SUMMARY

In a series of thirty-five carefully controlled cases infection has never occurred as a result of the approach to the round window niche through wide incision of the tympanic membrane. The round window membrane has not been ruptured, as evidenced by complete absence of any postoperative vestibular symptoms.

During the period of direct observation, possible only with magnified binocular vision and brilliant illumination, the graft has never failed to remain fixed in the round window niche. This represents an interval well beyond the time when normal tissue grafts might have failed to take, and had sloughed from their newly applied position.

Improvement in hearing fulfilling standards previously enumerated has been obtained following round window graft operations. The series has been small but has extended over a considerable period of time, three and a half years. When cases are selected according to the criteria listed above, and when the surgical procedure is carried

out as described, the operation is devoid of any serious immediate risk and in no instance has there been any late complication.

Limitation of applicability apparently lies only in the age group above 50 years, and to hearing losses greater than 50 decibels in the speech frequency range. It is hoped that the scope of the operation may be extended to the second decade age group after the hearing loss has reached a relatively static level.

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XXVI

CHRONIC STENOSIS OF THE LARYNX WITH SPECIAL CONSIDERATION OF SKIN GRAFTING*

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Chronic stenosis of the larynx continues to present some of the most difficult problems confronting the laryngologist. However, considerable progress has been made in the treatment of this condition in recent years and the prognosis has improved greatly. The full extent of this advance can be appreciated only when it is recalled that fifty years ago the outlook in such cases was generally considered hopeless, whereas at present with judicious persistent therapy a cure is to be anticipated in most instances.

The etiology of chronic stenosis of the larynx varies greatly. The condition may be the result of a congenital abnormality or it may result from accidental or surgical trauma. Radiation therapy often has a direct bearing on causation. Local or systemic disease, paralysis of the recurrent laryngeal nerves, lesions affecting the central nervous system and benign or malignant neoplasms of the larynx, may be responsible. The degree, character and extent of the stenosis may likewise differ. In fact, marked variance in all these features is encountered in different instances of the condition which have the same causative background. Lynch[1] called attention to the difference between the type of scar resulting from trauma and that caused by disease, noting that the scar tissue resulting from trauma followed by infection is far more extensive than that caused by disease. In LeJeune and Owens[2] experience, the most extensive stenoses resulted from perichondritis following trauma. At The Mayo Clinic the instances of cicatricial stenosis of the larynx most difficult from a therapeutic standpoint have been those caused by perichondritis following excessive irradiation.

The infectious granulomas, syphilis, tuberculosis, leprosy, blastomycosis, rhinoscleroma and others at times produce chronic stenosis

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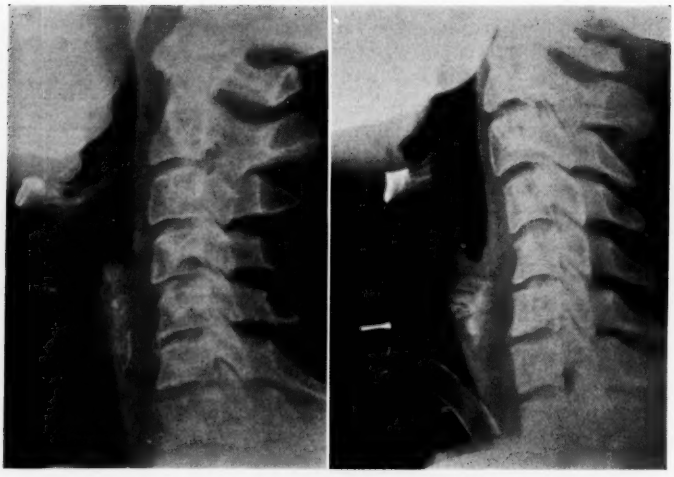
of the larynx. In these cases the obstruction may be caused by the active granulomatous process and the associated edema, or it may be caused by various complications and sequelae. Perichondritis with necrosis of the cartilage, sloughing and subsequent scarring, may bring about more or less permanent narrowing or even complete atresia of the laryngeal or tracheal lumen. Such changes are particularly prone to occur in syphilis, less frequently in tuberculosis, but they may be encountered in any of these granulomas. In Thomson's[3] experience, ulceration of tertiary syphilis of the larynx has been the most common cause of the condition. Chronic subglottic laryngitis at times causes marked stenosis. During the active stages of these diseases therapy must be directed toward control of the infectious process as well as relief of the respiratory obstruction.

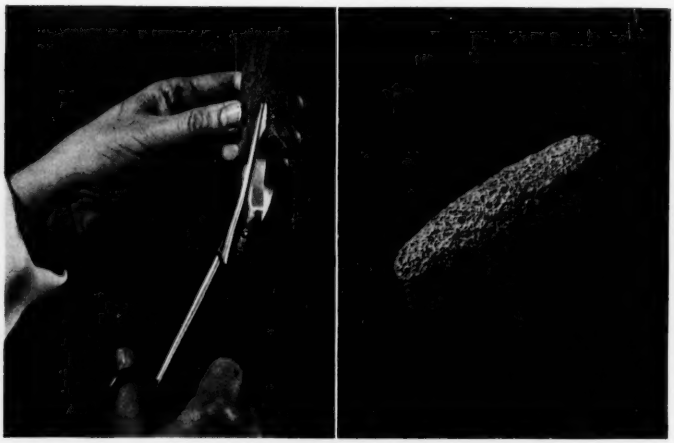
Treatment for chronic cicatricial stenosis of the larynx cannot as a rule be looked on as an emergency measure, but as Schmiegelow[4] stated, "The operative treatment of chronic laryngeal stenosis is to be compared with a cosmetic operation." Accordingly, therapy should be delayed until the patient is in the best possible general health and until it can be ascertained with reasonable certainty that the pathologic process responsible for the condition has been eradicated. Syphilis, tuberculosis or any other granulomatous, infectious or neoplastic process having a causative association must be controlled. Otherwise, these conditions may become activated by the trauma incident to treatment and undue local reaction with possible necrosis of the cartilages and sloughing may ensue. Systemic activation of tuberculosis is always a possibility in those cases in which this infection is responsible for the stenosis, and it is of the utmost importance that radical measures for correction of the narrowed lumen be delayed until prolonged observation provides convincing proof that the disease has been arrested. This statement also holds true concerning cases in which treatment of multiple papillomas or other benign or malignant neoplasms of the larynx has resulted in stenosis. Premature attempts at correction of the contracted air passage in these instances not only are doomed to certain failure but may result in grafting of neoplastic material on previously unaffected regions.

The type of treatment indicated in chronic stenosis of the larynx depends largely on the character of the stenosis and its extent, rather than on its cause. Accordingly, it is essential to determine accurately the exact situation, nature and extent of the obstruction so that the problem of therapy can be intelligently approached. In adults, examination by mirror alone often yields sufficient information, although direct or suspension laryngoscopy may be required. Roent-



(a) (b)

Fig. 1. (a) Lateral roentgenograms showing normal larynx and trachea on left; (b) on the right, fracture of larynx with marked stenosis.



(a) (b)

Fig. 2. (a) Paring airfoam rubber sponge to form a soft elastic core; (b) the core completed. This core may be used as a support for a skin graft within the larynx or it may be covered with one or more finger cots and anchored in the larynx to furnish continuous, gentle dilatation over a period of several weeks or months.

genographic studies, particularly with tomography, frequently are helpful (Figs. 1a and 1b). In children, direct laryngeal examination is necessary. When obstruction is complete, retrograde examination through the tracheal stoma many times will furnish additional information.

Improperly performed tracheotomy is a common cause of laryngeal stenosis, as is well known. Many times, particularly in small children for whom emergency tracheotomy has been necessary because of obstruction caused by streptococcic laryngotracheitis, diphtheria, the presence of a foreign body or some other acute laryngeal condition, decannulation is impossible, even after some months. Commonly, in such instances, the tracheal tube has been inserted directly into the larynx and its presence has prevented subsidence of the inflammatory process. Although simply placing the tube at a lower level often will be followed by spontaneous clearing of the stenosis, not infrequently marked scarring also is present in such cases and will necessitate additional treatment later. Many times perichondritis, resulting from the presence of a foreign body or attempts at removal of a foreign body, will cause persistent obstruction. The inflammatory process in the larynx in such instances usually will subside spontaneously on removal of the irritating factor, but it may be extremely slow in clearing and decannulation often may be delayed for months and at times for a year or more.

The profession often has been warned against opening the trachea through or above the first cartilaginous ring, and this advice should be heeded strictly in treating children. Concerning adults, however, the admonition is not pertinent, unless the tracheal stoma is to be permanent, in which event it should be situated as low in the neck as possible. For years it has been our practice in treating carcinoma of the larynx at the clinic to open the air passage through the lower border of the cricoid cartilage or the first tracheal ring, depending on the height at which the lesion is situated. This is done as a preliminary measure in all cases in which laryngectomy is necessary and also in those in which it is impossible to determine from the clinical examination whether or not laryngectomy will be required. Subsequently, the lesion is explored through a laryngofissure and if its extent necessitates it, laryngectomy is carried out. However, it is frequently feasible to remove the growth through the incision made during thyrotomy, but in our experience laryngeal stenosis never has resulted from performance of high tracheotomy in such instances.

Fig. 3. Incision in the neck at the end of ten days following open operation for stenosis of the larynx. Note complete healing of the soft tissues and the very slight inflammatory reaction (the discoloration is the result of application of gentian violet solution).

Fig. 4. Incision in the neck at time of removal of original dressing, ten days following open operation for cicatricial stenosis of the larynx. The silver wires anchoring the rubber sponge and skin graft within the larynx were left long in order to hold the external dressing in place. In the illustration these wires have just been divided and will be removed externally before withdrawing the rubber sponge from the larynx.

Fig. 5. Postoperative scarring of the neck of patient shown in Figs. 7 and 8 two and one-half years later.

Bouginage is the procedure most commonly employed in the treatment of chronic cicatricial stenosis of the larynx. In cases in which the scarring forms a thin diaphragm, this form of therapy is very effective. It is useful also in preventing readhesion of the margins of divided congenital webs, although when such anomalies are more than a few millimeters in thickness, maintenance of separation in this manner may be impossible. As a rule, it is futile to employ bouginage in instances of extensive cicatricial stenosis of the larynx. The procedure usually must be carried out at frequent intervals over a long period, the frequency of repetition depending on the amount of local reaction induced. Trauma sufficient to cause bleeding always should be avoided, otherwise increased scarring may ensue. For many patients seen in The Mayo Clinic, bouginage has not proved effective in relieving the stenosis and it has been necessary to carry out more radical measures later. However, it should be stated that since most of our patients live at some distance from the clinic, the prolonged close observation necessary for effective bouginage has not been possible. We have had no experience with the electrically heated bougies described by Dean[5] and Johnson.[6]

Constant elastic pressure has been much used in the field of plastic surgery to correct cicatricial stenosis of the nostrils, external auditory canals, nasopharynx and other structures. It tends to produce thinning-out, softening and increased pliability of even dense scar tissue, and if properly used over a long period often will completely overcome such narrowing. Very soft rubber tubes are employed for the purpose, being replaced at intervals with successively larger tubes. The utmost care during insertion of these tubes is essential to avoid trauma which might induce sufficient inflammatory reaction to necessitate their removal. For the same reason, undue pressure must be obviated by carefully graduating the size of the tubes used. Until the dilatation has been carried to a certain point, stenosis will recur very promptly if the dilators are left out for even a few hours. As a matter of fact, there is a tendency for a certain amount of contracture to take place for months and, accordingly, it is necessary to overdilate and in some instances to use the dilators for brief periods at later intervals. This same principle is frequently employed in the treatment of cicatricial stenosis of the larynx. Even though only a very narrow tract remains in this structure, it is usually possible to insert a rubber dilator, which may at first be only a filiform or a piece of ureteral catheter. Continuous wearing of this dilator by the patient and replacement of it with successively larger tubes over a long period may of itself completely overcome

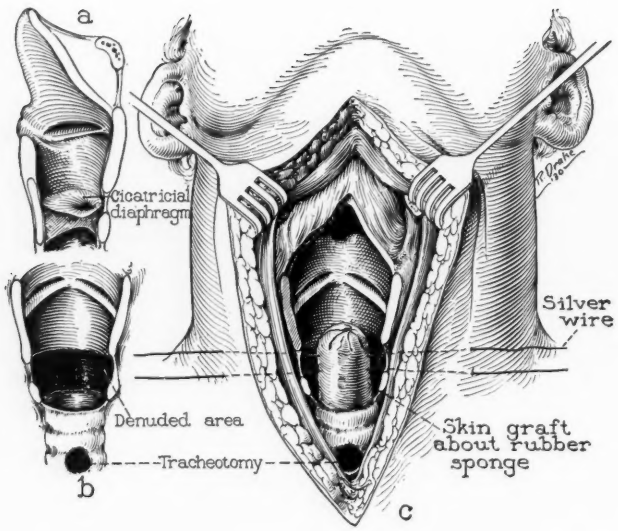


Fig. 6. (a) Cicatricial stenosis of the subglottic portion of the larynx; (b) the cicatrical diaphragm has been excised and the trachea opened; (c) a thin skin graft has been inserted about an elastic core which is anchored in place by means of a silver wire transfixing the front of the neck and the larynx.

the stenosis. Iglauer's[7] use of a folded rubber tube for this purpose is an ingenious and simple application of the procedure. In this situation even greater care, gentleness and patience are essential than are required in treating more accessible strictures, for attempts to hasten the dilatation by using tubes that are excessively large or hard may induce sloughing or necrosis of the cartilage. Should it become necessary to discontinue the treatment because of undue local reaction, at least several months are likely to elapse before the inflammatory process will clear sufficiently to permit of further therapy.

These tubes can at times be inserted in adult patients by means of indirect laryngoscopy and local anesthesia, but more frequently laryngeal suspension and general anesthesia are employed. In children the latter combination is essential. The core molds of Jackson[8] are essentially the same as the rubber tubes, but because of their shape they tend to remain in position better. Both, however, must

be anchored in place. In the case of the tubes, a heavy silk ligature through the lower end is tied directly to the flange of the tracheal cannula. As it is at times employed, a screw inserted through the front of the neck into the dilator definitely renders the apparatus self-retaining. Whether a closed or an open dilator is to be used may be entirely a matter of choice or it may depend on the conditions present in the individual case. Each dilator possesses certain advantages. An open tube, if it is of sufficient size, will permit of breathing in the normal manner and is usually preferred by the patient. Schmiegelow frequently removes the tracheal cannula on inserting a dilating tube in adults, so that respiration would be carried on entirely through this tube. Unless the height of an open tube is carefully adjusted, however, liquids and particles of food may be aspirated through it. In most instances I have used as dilators very soft tubes or a core of airfoam rubber sponge covered with two rubber finger cots; a silk ligature closes the open lower end of these cots and anchors the dilator to the tracheal cannula (Figs. 2a and 2b). The length of time it is necessary for the patient to wear these dilators varies greatly. It can often be shortened materially by incising the margins of the narrowed lumen so that a larger tube can be inserted primarily; this procedure is likely to be successful when the stenosis is caused by a comparatively thin cicatricial or congenital diaphragm. In most instances in which the scarring is extensive, open operation is decidedly preferable: it is safer, less trying to the patient, requires less supervision and accomplishes the desired result in a shorter time than other types of treatment (Fig. 3).

Until recent years, laryngologists generally felt that an open operation for the correction of cicatricial stenosis of the larynx should be carried out only as a last resort. Many still insist that the possibilities of all conservative measures be exhausted before considering radical surgery. However, increasing experience demonstrates that in many cases of marked cicatricial stenosis an open operation offers the best and at times the only possibility of restoring the lumen, and that it might better be resorted to first than last. Obviously the patency of the semirigid larynx and the trachea can be brought about only by removing the scar tissue or the distorted or thickened cartilage that is producing the obstruction, and by furnishing a new lining membrane.

Schmiegelow in his Semon lecture of 1937 presented a series of cases of cicatricial stenosis of the larynx in which excellent results were obtained merely by excising the scar tissue through a laryngofissure and inserting a rubber tube dilator which was left in position

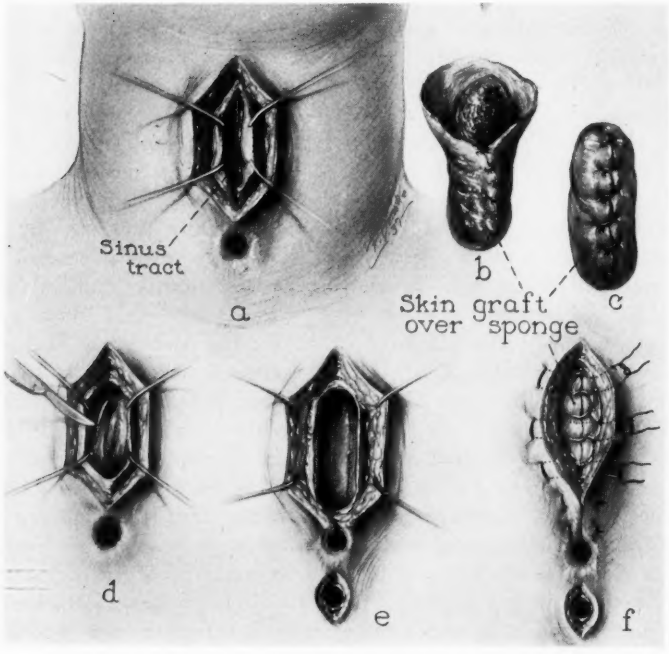


Fig. 7. (a) Tiny tract remaining within the larynx of a patient who had marked stenosis resulting from perichondritis; (b, c) airfoam rubber sponge covered with skin graft ready for insertion into restored laryngeal lumen; (d) inadequate laryngeal lumen resulting from excision of cicatrix; (e) adequate lumen restored by coring out the thickened cartilage. The site of the tracheal stoma which was situated at the inferior border of the stenosis has been lowered; (f) skin graft inserted in larynx and incision in neck partially closed.

for from a few weeks to eleven months. In plastic procedures performed about the surface of the body and within the mouth, nose, vagina and urethra, epithelization of freshly denuded portions can be effected most rapidly and with the least contracture by means of skin grafts. If cut thin, these grafts grow very well in infected regions and they produce no trouble later. In the light of experience with such grafts in such regions, their use in relining the larynx and trachea seems definitely indicated, since the healing period is thereby shortened and there is less tendency for grafted surfaces to contract.

Use of skin grafts in the treatment of stenosis of the larynx, introduced by Arbuckle[9] in 1930, marks a distinct advance in the therapy for this condition. Since then numerous reports on the use of such grafts have appeared in the literature, notably those by Negus,[10] LeJeune and Owens,[2] Foster[11] and Figi,[12] and the results have been uniformly satisfactory (Fig. 6, a, b and c).

At The Mayo Clinic this operation is performed on adult patients under cervical block anesthesia; children require a general anesthetic. The larynx and, if necessary, the upper portion of the trachea, are opened in the midline and the scar tissue is completely excised to restore the lumen. If thickening or distortion of the cartilage is present, attention also must be directed to it.

Skin grafts to serve as lining are best obtained from a surface free from hair, and in preparing them, they should be cut sufficiently thin so that any hair follicles which might happen to be present will be left behind. In spite of the surgeon's efforts in this regard, however, a few hairs frequently are transplanted. Although these are not desirable, they cause no particular trouble unless they are present in excessive numbers. Most satisfactory grafts may be obtained with the new dermatome of Padgett or Hood, which cuts them of uniform thickness. In our more recent operations the graft has been held in place by wrapping it about a core of airfoam rubber sponge which has been made somewhat larger than the laryngeal lumen, so that it will exert light constant pressure against the cartilaginous wall. Fine silk sutures prevent the graft from slipping off the sponge. The core is immobilized within the larynx by transfixing it with one or two silver wires threaded on a long straight needle and passed directly through the skin and the thyroid cartilage, after the method of Schmiegelow. These wires may be cut off short enough so that the ends retract beneath the skin, as Schmiegelow has suggested, but I have at times found it advantageous to leave them sufficiently long that they may be brought across the front of the neck to hold the dressing in place (Fig. 4).

Provided that there is no undue reaction, the rubber sponge supporting the skin graft is left in place for ten days. It may be removed at the end of a week, but the graft is less firmly adherent after this shorter period of time and is more readily displaced than it would be at the end of ten days. Leaving it in place longer than ten days will do no harm. At the end of this time the wound in the neck is usually healed and the sponge can be removed by grasping it with laryngeal forceps after exposing the larynx with a direct laryngoscope or with the suspension apparatus. The silver wire

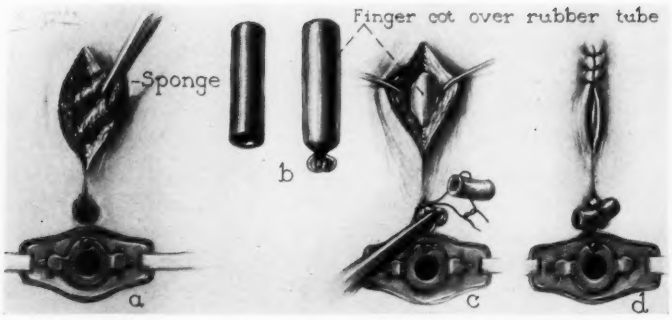


Fig. 8. (a) Rubber sponge being removed at the end of ten days through the partially reopened incision in the neck; more frequently this is taken out through the mouth; (b) soft rubber tube dilator covered with finger cot; (c) the dilator has been inserted into the larynx to replace the rubber sponge and in this instance was anchored in position by means of a silk ligature brought out through the original tracheal opening and tied about a rubber tube; (d) the incision in the soft tissues is being closed over the dilator.

anchoring it in place is pliable enough so that it can be drawn out with the sponge, but it has seemed preferable first to remove the wire externally because of the possibility of traumatizing the graft as it is pulled up through the larynx. The skin graft usually is perfectly successful unless the sponge about which it is wrapped becomes displaced. In fact, I have never seen such a graft fail to grow satisfactorily.

In cases in which there has been no appreciable loss or distortion of cartilage it may be unnecessary to insert a laryngeal dilator at this time, but it is advisable to do so in the majority of instances, otherwise the marked tendency for thin skin grafts to contract and the lack of rigidity of the cartilage in many of these instances may result in recurrence of the stenosis. I prefer to insert a dilator similar to the core used to support the skin graft. The elasticity of this dilator permits of its conforming to the irregularities of the interior of the airway, yet it maintains constant gentle dilatation. It is worn for from a few weeks to several months or at times even longer, depending on the individual case. It has seemed preferable to leave the dilator in position longer than is necessary, rather than not long enough. In the cases in which patients have been treated to date,

it has not been necessary to replace the dilator or to use bouginage subsequently. In Schmiegelow's cases, in all of which the patients were treated without skin grafts, the rubber tube was left in place in the larynx for an average of six weeks. In three instances it was necessary to leave the tube in place for six, nine and eleven months, respectively. On removing the tube through the mouth, if the laryngeal opening was not sufficiently large, another laryngofissure was made and insertion of a larger drain done. In one case involving severe syphilitic perichondritic stenosis, the operation was repeated five times.

Most patients for whom we have found it necessary to carry out external operations and skin grafting for stenosis of the larynx have been in the second and third decades of life. The oldest patient was 59 years of age, the youngest 13 years of age. I should not hesitate to perform the procedure for small children, however, should it appear indicated. Barring severe infection, it does not seem likely that interference with development of the laryngeal cartilages would result in such instances, provided the growth of the cartilages had not been arrested previously by the stenotic process. This procedure seems decidedly preferable to laryngostomy, which has been so commonly employed in the past. The prolonged packing necessary after laryngostomy is tedious and painful and close supervision is essential throughout the treatment. On the other hand, once the rubber dilator is in place after the former operation, very little attention is required. Obviously, it is much easier to maintain constant elastic pressure within a closed tube than in one that has been laid open.

A matter of serious moment in instances of cicatricial stenosis of the larynx is possible loss of the sphincteric action of the superior laryngeal orifice. Fortunately, such a complication is only rarely encountered. In severe postdiphtheritic stenosis which afflicted a boy 15 years of age, as observed by Nager,[13] the airway was reestablished by dissecting out the scar tissue and relining the larynx with a skin graft. However, following this procedure the patient had difficulty in eating and drinking, because anything taken by mouth tended to pass directly down into the larynx and trachea. Continuance of this difficulty necessitated reproducing the stenosis.

The importance of growth in overcoming stenosis of the larynx occurring in childhood has frequently been stressed in the literature. Unquestionably, forcing the child to breathe through the larynx is of marked benefit in certain types of stenosis. In many instances of dense cicatricial contracture, however, particularly in those result-

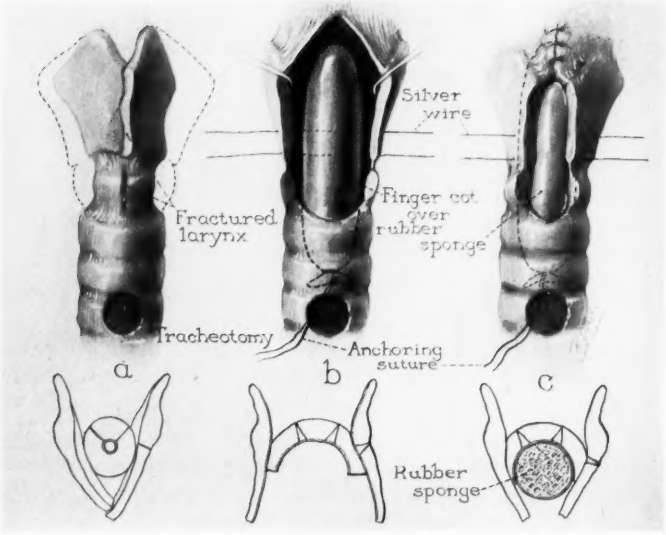


Fig. 9. (a) Fracture of larynx with stenosis; note lateral compression of thyroid cartilage and concentric contracture of cricoid; dotted lines show approximate outline of normal larynx; the diagram below is a cross section to show degree of stenosis; (b) larynx opened in midline and rubber sponge covered with finger cots inserted to serve as dilator; dilator anchored in place by means of silver wires; ligature at times used to anchor dilator to tracheal cannula is also shown; the diagram below shows incisions through cricoid and left wing of thyroid cartilage to overcome contracture; (c) larynx closed over dilator; note gaping between edges of thyroid and cricoid cartilages caused by dilator within the larynx.

ing from intensive irradiation therapy, the growth of the larynx has been arrested and no improvement will follow even prolonged enforced laryngeal respiration. At the clinic our observation in this regard is in keeping with that of Negus. Various methods of treatment have been carried out for such patients, but the only procedure that has proved of real benefit has been excision of the scar tissue and thickened cartilage through a laryngofissure, and prolonged use of a rubber dilator.

In one case in which stenosis of the larynx had resulted from perichondritis following treatment for multiple papillomas, extreme

thickening of the cricoid cartilage was present. The laryngeal lumen was reduced to a minute tract that scarcely admitted a small probe. The stenosis had been caused largely by the thickness of the cartilage rather than by cicatrix, and removal of the latter produced only slight enlargement of the narrow airway. However, coring out the inner portion of the thickened cartilage created a lumen of approximately normal size, leaving only a cartilaginous shell that was so thin and soft that it appeared questionable whether it would furnish sufficient support to maintain an adequate air tract if the lining were restored. Doubt arose also concerning the likelihood of a skin graft's adhering directly to the surface of this cartilage, since it was entirely denuded of fibrous tissue. In spite of apprehension in this regard, the graft grew perfectly and the cartilaginous support has proved to be ample (Figs. 7a, b, c, d, e, f; 8a, b, c, d, and 5).

Fracture of the cartilages of the larynx may render treatment of stenosis of this structure much more difficult than it might otherwise be. Severe respiratory obstruction often develops promptly after such an injury, especially if there is much displacement of the fragments of cartilage. Tracheotomy usually is necessary, although it may not be required before the end of several days or even a week or two. If the fracture is recognized early, a soft rubber dilator inserted into the stenosed larynx by means of direct or suspension laryngoscopy may forestall marked cicatricial narrowing later.

Frequently, however, patients do not come under the observation of a competent laryngologist until several weeks or months following the injury, at a time when persistence of the obstruction has made it obvious that the process will not clear spontaneously. Firm healing of the displaced cartilage fragments may be present and this is usually complicated by cicatricial stenosis. There may be marked lateral narrowing of the thyroid cartilage, also concentric contraction of the cricoid cartilage. In such cases, following laryngofissure and excision of the scarring within the larynx, it is at times necessary to make multiple incisions through the distorted cartilage from the external surface inward in order to overcome the spring of the distorted cartilage and permit of replacing the parts in approximately normal position. A skin graft is then inserted in the usual manner and the patient wears an elastic dilator for a prolonged period. Even though there may have been considerable loss of cartilage and even though a large segment of the cricoid ring has been destroyed, restoration of the normal airway is possible in such a manner. In one such instance, involving a male patient 22 years old, the injury occurred in an automobile accident, and approxi-

mately one-third of the cricoid cartilage had been lost. In spite of this extensive damage, the ends of the remaining portion of the cricoid cartilage had drawn together and were firmly united without overlapping. Dividing the cartilage along the line of union and at several additional points permitted of opening up the ring. Long continued wearing of a dilator by the patient resulted in restoration of a satisfactory lumen (Fig. 9a, b, c).

Generally speaking, skin flaps have comparatively little place in the treatment of stenosis of the larynx because of their excessive bulk. It is practically impossible to obtain a flap sufficiently thin to serve as a lining for the larynx and have it remain viable in the presence of the infection encountered in the laryngeal region. However, at times, extensive destruction of tissue over the front of the neck with a large perforation into the larynx or trachea, caused by excessive irradiation or by removal of a neoplasm, is encountered. In such instances a lined pedicled flap may afford the only possibility of reconstruction.

SUMMARY AND CONCLUSIONS

1. Treatment for chronic stenosis of the larynx should be delayed until the patient is in the best possible general condition and until any local inflammatory reaction has entirely cleared.

2. The type of treatment indicated in a given instance depends largely on the character and extent of the stenosis rather than on its cause.

3. Bouginage, the most commonly employed procedure in the treatment of stenosis of the larynx, has in our experience at The Mayo Clinic been one of the least effective procedures.

4. Elastic pressure applied by means of soft rubber dilators is one of the most satisfactory methods of treating chronic stenosis.

5. Laryngofissure with excision of the scarring and any thickened or distorted cartilage, followed by skin grafting, is the most effective method of dealing with chronic cicatricial stenosis and often will restore a satisfactory laryngeal lumen after other methods of treatment have failed.

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XXVII

ADVANTAGE OF MIXED BONE AND CARTILAGE GRAFTS IN CORRECTION OF SADDLE NOSE AND OTHER DEPRESSED DEFORMITIES OF THE DORSUM

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BALTIMORE

All those familiar with the progress made in correction of saddle nose and other depressed deformities of the nasal dorsum during the past three decades recall how various foreign substances have been placed beneath the skin of the nose, in an effort to restore its shape and form. Thus, paraffin, celluloid and ivory have had their day, and after fair trial have, for valid reasons, been found wanting.

Paraffin, softening as it does during hot weather, tends to migrate from the site of implantation into the surrounding tissues and, so we found, created in many instances a far greater deformity than originally existed. The frequent necessity, therefore, for removal of the so-called paraffinoma has placed this substance definitely in the discard.

Celluloid, according to New,[1] first used by two Vienna surgeons, Föderl (1903) and Kuchier (1908), was subsequently employed in 1916 by two English surgeons, Higgins and Thompson, in three cases for correction of saddle nose. New, in animal experiments, placed celluloid beneath the skin, over the nose of six dogs. In four of these primary union was obtained, but in the other two infection necessitated removal of the implants. He reported having subsequently employed celluloid in five patients for correction of saddle nose. All healed satisfactorily and, after a year's observation, no trouble had ensued. In answer to a recent inquiry, however, New[2] writes, "I had several pieces of celluloid come out after I used it years ago. I gave up using all types of foreign material. We are now using bone or cartilage implants only."

Ivory, of all substances used for implants, seems longest to have retained popularity, and is still used by a number of surgeons. It is a well established fact that the human tissues will not indefinitely

and under all conditions tolerate the presence of any foreign substance, and it makes one wonder just why the use of ivory is not also completely discontinued. My own experience and that of other surgeons would appear to justify the plea against its further use. Aufricht[2] wrote he had used it in 50 or 60 cases, and remembers in only one case where removal was necessary because of infection. He reports that in a few cases of his own and other surgeons, however, he "had to remove the ivory for aesthetic reasons (usually they were too large) and substitute them with smaller ones." Further, he states, "I discontinued its use about eleven or twelve years ago because, on general principles, I do prefer auto-transplants in the nose whenever possible." In a more recent letter, he writes, "As an interesting coincidence, since my last letter, a patient with saddle nose for whom I implanted ivory in May, 1926, returned complaining that the implant had sunk toward the tip of the nose during the past year. No inflammatory process was present, but the lower end of the ivory was working its way through the skin, making its removal necessary." I, myself, have had similar experiences in two cases in which ivory had been inserted by other surgeons.

Sallinger,[4] in a recent report of 65 cases, said that in one case the ivory implant inserted sixteen years before is still satisfactory. Sallinger is satisfied that ivory is safe and will yield satisfactory results if properly done, in spite of the fact that in five of his cases infection necessitated removal of the implant—three 48 hours after operation, and in two cases "after exposure of the nose to sub-zero weather," one of these four days after operation and the other a full year following operation. These experiences certainly offer little argument for the safety of ivory as an implant. In a second group of five cases he states that "an intolerance of the tissues seemed to exist. This condition was manifested by persisted redness of the overlying skin, and after a variable length of time a fistula formed at the lower extremity of the implant, or within the vestibule, making removal of the ivory imperative." As it is manifestly impossible to determine beforehand when such intolerance of the tissue for foreign substance may exist, why take chances by making use of it at any time, when we have in the auto-implant a safety which has been proven in the work of so large a number of surgeons?

The two main reasons offered for the continued use of ivory by its advocates are that it eliminates the need for the double operation required in obtaining rib sections with possible danger of infection of the pleural cavity, and the fact that patients refuse to submit to removal of the rib for that purpose. Neither of these reasons

seem to be of sufficient importance to warrant the substitution of ivory for the auto-implant. In several hundred private and clinic cases done by the essayist during the past twenty-five years, not one chest complication occurred, and not in one instance has a patient refused to submit to the removal of a piece of rib after having been told the reasons for and results of our method. Nor have I been obliged to remove a single implant because of infection during the past twenty-two years, and only two prior to that time.

Murphy,[5, 6] over a quarter of a century ago, in his epoch making studies and experiments with bone graft, showed that bone transplanted in the same individual when contacted with living osteogenetic bone, always becomes united to the living bone and acts as a scaffolding for the reproduction of new bone of the same size and shape as the transplanted fragment, if perfect asepsis has been attained. Does one need better proof of the safety and advantage of autogenous bone grafts? Murphy's results have been borne out practically 100 per cent in our use of the mixed bone and cartilage graft.

Some years ago a section from the anterior border of the tibia was given a trial, but found unsatisfactory. Such dense brittle bone was indeed difficult to shape, and it soon became apparent that it did not unite with the underlying nasal bone. Without such union, proper nourishment of the graft is impossible, and it remains a more or less movable foreign body beneath the skin, slowly undergoing absorption, with a gradual return of the deformity in four or five years. In addition to this, removal of a section from the anterior border of the tibia, the actual keystone of its arch, so weakens its shaft that fracture occurs from very trivial trauma. Such fracture of the tibia occurred in two of my very early cases, one within a year and the other about two years after operation.

For some years the vast majority of plastic surgeons have been making use of costal cartilage alone in the correction of saddle nose, and many still continue to do so. These surgeons feel that cartilage is more resistant to infection than bone, is less likely to be absorbed and more easily given the desired shape than bone. In our experience, cartilage, which never unites with bone, remains fixed by connective tissue alone to the underlying bones and the overlying skin. It is, therefore, always more or less movable under the skin, and after several years some absorption always takes place, with a certain amount of irregularity developing along the dorsum of the nose, unless the perichondrium is left on the upper surface of the graft.

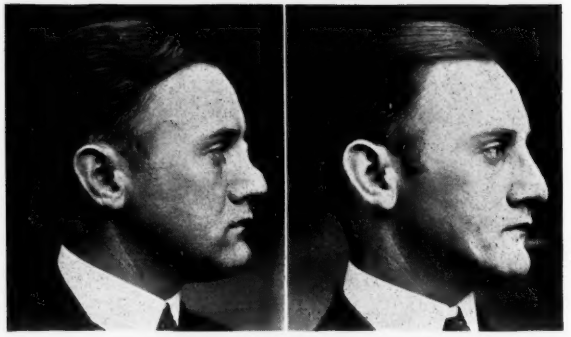
But, since a strip of cartilage of sufficient length for a complete graft tends to curve upward toward its perichondrial surface, operators, using cartilage alone, remove the perichondrium entirely to prevent such curving.

About the year 1912, after having had the privilege of witnessing an operation of Carter, in which rib bone alone was used to correct a saddle nose, we began some studies and experiments, with the idea of determining whether firm union might always be accomplished between such graft and the underlying bone. Our results after a time convinced us that, when the periosteum is completely removed from the top of the nasal bones as well as from the apposing surface of the rib section, union between them is firmly established, just as occurs when two ends of a fractured long bone are held in apposition by a splint. This was further confirmed a few years later on two occasions when patients, following faultily placed bone grafts, were referred to us for correction. In these cases, because of inadequate splinting, we found the axis of the grafts had become displaced from the midline of the nasal dorsum. Removal of these grafts, because of the firm union, could only be accomplished with a sharp chisel and hammer. We were now satisfied that rib bone, which biologically so closely resembles the nasal bones, with their two compact layers separated by a narrow space of diploic structure, would unite with the top of the nose and become an integral part of its bony framework, provided it was held firmly in contact by a suitable splint for a sufficient time to establish such union.

We found, however, in the use of bone alone, the contact of its lower end against the soft tissues of the nasal tip offered a problem. The lower end of the bone, the edges of which are always more or less sharp, caused a certain amount of irritation, and in several instances had to be shortened somewhat by a secondary operation, to avoid perforation at the tip.

By 1915, we had determined that a graft removed from the osseo-cartilaginous junction of the seventh or eighth rib, so that the graft when finished would consist of approximately three-fifths bone to contact the nasal bones, and two-fifths cartilage to be placed over the cartilaginous portion of the nose, would furnish an ideal graft embodying all the advantages of either bone or cartilage, with none of the disadvantages of either when alone employed. Periosteum and perichondrium are always left on the upper surface of our graft, to guard against the development of future irregularities along the dorsum. In this mixed type of graft we had never ob-

Congenital saddle nose in which entire thickness of rib bone and cartilage was utilized to correct the deformity.



Saddle nose with double hump induced by curving of a cartilaginous graft, inserted in another city. Curved cartilage removed and mixed bone and cartilage graft inserted at a single operation, with result shown herewith.

served any tendency of the cartilage to curve upward toward the perichondrial surface until early in 1939, when a man operated on in 1926 returned with the lower beveled end of the cartilage curved upward. I have no doubt that this condition arose only because the lower beveled end of the cartilage was made too thin. We have since then, however, been removing the perichondrium from the lower third of the cartilaginous portion of the graft only, and I have no doubt that we shall in the future not be troubled by such an occurrence.

As far as difficulty in shaping of graft is concerned, we have found that by using the sharp chisel and bone shears for the bone and the knife for the cartilaginous portion, the graft may be given any shape necessary to fill in the depression of the dorsum with as much ease as when cartilage alone is used for a graft.

To obtain a lasting ideal result, the upper surface of the graft must always extend in a continuous line from the nasal notch above to the tip below, and be so shaped that the nasal notch will not be effaced. To accomplish this it is obvious that the shaping of the graft must be entirely done from its lower surface. In case the depression is confined to the cartilaginous portion of the nose, as occurs after too extensive removal of cartilage in submucous resection and septal abscess, we remove the inner table of the bony portion of the graft along with the entire diploic structure, and so shape the thicker cartilaginous portion of the graft to fill in the depression. That the dorsum nasi should not appear too wide or too flat the upper end of the graft must be narrow, varying in width from one-eighth to three-sixteenths of an inch, while the lower cartilaginous end should vary from three-sixteenths to one quarter inch, according to the size and width of the nose to be corrected.

The upper end of the graft should be beveled from its under surface, beginning about three-sixteenths of an inch from its upper edge where it contacts the frontal notch. It is desirable also to make, with the elevator, a pocket beneath the periosteum, into which the upper end of the graft is inserted. The upper end of the graft is thus better held in place and firm union at the frontal notch is assured.

The lower cartilaginous end of the graft must likewise be beveled, from the under surface near the tip, and rest in a pocket extending to the fleshy portion of the tip, but not so far down, however, as to make undue pressure against the skin overlying the

nasal tip. Excessive pressure against the skin in this situation might be followed by necrosis of the skin.

In some cases of saddle nose where the cartilaginous septum has been completely destroyed, it is necessary to insert between the two layers of muco-perichondrium a strip of cartilage extending from the anterior nasal spine below to the lower surface of the dorsal graft just above the tip. One decided advantage, however, in the mixed bone and cartilage graft is, that by the firm union of the bony portion with the nasal bone above, the lower end of the graft is prevented from dipping downward, so that an intra-septal graft becomes necessary only where the contraction which follows a total destruction of the septal support draws the tip downward too greatly.

For complete details of technique in obtaining the rib graft, preparing and placing it, you are referred to a monograph of the essayist[7] read before the Southern Medical Association, November, 1936.

SUMMARY AND CONCLUSIONS

We have in the autogenous graft from the rib an implant which if handled under strict asepsis, is properly shaped, and held in place beneath the skin of the nasal dorsum, gives us assurance that it will be permanently tolerated by the tissues under all normal conditions. While this holds true for the costal cartilage implants, we are certain from our own experience that in time, when cartilage alone is used, a certain amount of absorption takes place, with a recurrence of some deformity. We are, however, convinced that in the mixed bone and cartilage graft from the seventh or eighth rib, we have the ideal implant, and that when firm union of the rib bone is established with the bony nasal dorsum, the graft will live without absorption and return of any deformity for the life of the patient. We know, furthermore, that if our technique is meticulously carried out, infection of a bone graft is no more likely than that of cartilage.

In conclusion, we hope that the time is not far distant when all surgeons will so thoroughly realize the advantage and safety of the autogenous graft from the rib, that ivory or any other foreign substance will no longer have a place in this work, and that the advantages of mixed bone and cartilage graft over that of cartilage alone, will be recognized and employed by surgeons interested in rhinoplasty.

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XXVIII

CARCINOMA OF THE LARYNX

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The treatment of intrinsic laryngeal carcinoma usually yields excellent end results. It is reported that 60 per cent of the lesions requiring total laryngectomy and 80 per cent of the more superficial ones amenable to laryngofissure are living and well after five year periods. On the other hand, the treatment of extrinsic carcinoma of the larynx is most discouraging, five-year cures being the exception and, unfortunately, a very large number of laryngeal neoplasms fall into this group. The purpose of this communication is to discuss the results of a critical study of one hundred extrinsic laryngeal neoplasms and to report the end results of thirty-one surgically treated intrinsic laryngeal malignancies.

EXTRINSIC LARYNGEAL CARCINOMA

The extrinsic lesions have been divided into two groups:

1. Primary extrinsic laryngeal carcinoma.

2. Secondary extrinsic carcinoma resulting from extension or metastasis of primary intrinsic growth.

PRIMARY EXTRINSIC LARYNGEAL CARCINOMA

Fifty-seven of the one hundred lesions studied were primarily extrinsic. They arose from an epiglottis, pyriform sinus, aryepiglottic fold, or from an arytenoid. The anatomic site of these neoplasms influenced both their cellular structure and their clinical behavior, but as the more important cytologic and clinical characteristics were similar they will be discussed collectively.

When first seen these neoplasms were all advanced, were usually bulky and had large, cervical metastatic nodes. Histologically they were usually epidermoid in character and often highly anaplastic. Although anaplastic, frequently they did not tend to infiltrate, being papillary in character, so that pain was a late symptom noted only after secondary infection had set in and, because of their anatomic

location, early voice changes were the exception. The patients usually sought medical advice only because they had noted enlarged glands in the neck, so that the disease was far advanced when first seen.

All these patients were treated by external irradiation, either with the radium bomb or x-ray machine. A fractional technic was followed, total doses of 6000 to 7000 R. U. or 200 to 250,000 M. H. being employed. Cytologically, these neoplasms often were radiosensitive, being anaplastic and non-infiltrating, so that excellent primary regression was often seen, but the end results were uniformly disappointing.

SECONDARY EXTRINSIC LARYNGEAL CARCINOMA

Forty-three of the 100 lesions studied were secondarily extrinsic. As in the primary extrinsic group, the anatomic origin of these neoplasms modified both the cytology and clinical behavior, but as they were also far advanced, they too will be discussed as a group. As a rule these neoplasms were less bulky than the primary extrinsic ones. Cervical adenopathy was often present. Histologically, scirrhous infiltrating epidermoid carcinoma was usually encountered. Early voice change was the rule and dyspnea occurred frequently.

These patients were also treated by fractional external irradiation. Good primary regression, however, was seldom seen as these lesions were infiltrating and hence were radio resistant. The end results were bad, the uncontrolled disease making rapid progress.

The chief interest in this latter group was why an early, intrinsic, usually curable lesion was allowed to progress until it became advanced, extrinsic, and fatal. Four factors were found to be common denominators and, in the order of their importance, they were:

1. Failure of the patient to realize the significance of persistent aphonia. Hoarseness lasting months and even years had been ignored until large cervical nodes were evident, or until posterior invasion had resulted in pronounced dysphagia. Many of these tragedies might have been avoided through lay education, and while such programs have been undertaken, much remains to be hoped for.

2. Failure of the physician to recognize early, obvious pathology. The general practitioner was not always sufficiently impressed by a history of persistent aphonia, and being inexpert with the laryngeal mirror, had overlooked early lesions.

3. Overhanging, infantile type of epiglottis. This type of epiglottis obscured indirect view of the anterior commissure and, as direct examination had not been undertaken, small, intrinsic, anterior commissure lesions had not been discovered until extralaryngeal structures were involved.

4. Subglottic growth. Again direct laryngoscopy had not been performed and lesions which might have beeen amenable to laryngeal surgery became inoperable because they were hidden below the vocal cords and had escaped detection by indirect examination.

It should be borne in mind that even in skilled hands the laryngeal mirror has shortcomings. Carcinoma hidden under the epiglottis or in the subglottic space may easily escape unnoticed if direct laryngoscopy is neglected. Hoarseness persisting over a period of weeks should be investigated with the direct laryngoscope and biopsies should be taken from suspicious areas if the tragedies of extrinsic laryngeal carcinoma are to be reduced.

INTRINSIC LARYNGEAL CARCINOMA

The results of the surgical treatment of thirty-one cases of intrinsic carcinoma have been summarized in the accompanying table.

In this series of thirty-one intrinsic lesions there were four instances of recurrence or metastasis, and it is interesting to note that in two of these four, highly anaplastic lesions had been treated surgically. In the instance of recurrence after fissure, the initial lesion, although highly anaplastic, had not infiltrated, so that a better result might have been obtained had irradiation been employed instead of surgery, as excellent response to x-ray or radium may be expected in the presence of non-infiltrating lesions with movable cords. Such a lesion may even be differentiated, but the clinical interpretation of infiltration evidenced by the motility of the vocal cords is a more reliable indication of cellular response to irradiation therapy than is the cytology of the neoplasm. It is true that the growth was anatomically favorable for laryngofissure, but its cytology may have caused it to be biologically inoperable.

On the other hand, the anaplastic carcinoma treated by total laryngectomy offered no alternative for surgery. This lesion had infiltrated and caused fixation so that the response to irradiation would have been poor.

An infiltrating lesion may even be differentiated, but again the motility of the cords has proven to be a more satisfactory guide than the histopathologic picture. Furthermore, this lesion had subglottic extension and subglottic lesions are resistant to irradiation, good response seldom being seen.

Ten of the cases treated surgically had had adequate irradiation therapy but had failed to respond. Post-irradiation surgery was undertaken only after the epidermite and epithelite had subsided, but in spite of this precaution, healing was delayed and secondary closures were the rule. However, the ten cases demonstrate that even adequate amounts of irradiation, up to 6000 to 7000 R. U., do not contraindicate surgical intervention.

Concerning surgical technic, the narrow field, single, midline incision was employed for laryngectomy. Adequate exposure was obtained by retracting first one side and then the other. It was felt that the narrow field technic had two distinct advantages. First, the hyoid muscles were preserved and were used to reinforce the closure of the pharynx, and second, the blood supply of the soft tissus of the neck was not greatly interferred with so that the danger of postoperative sloughing was minimized. The anesthesia employed was a combination of local infiltration and intravenous morphine. The use of morphine in laryngeal surgery is unorthodox, but in this series it was employed without side reactions or complications. It was slowly injected at the time that the trachea was opened and the amounts used were never sufficient to depress the cough reflex too deeply.

CONCLUSIONS

One hundred cases of extrinsic laryngeal carcinoma have been reviewed and an attempt made to establish why primary intrinsic disease became secondary extrinsic disease.

The end results of thirty-one surgically treated intrinsic carcinoma of the larynx have been reported. In this group thirteen of the fourteen patients who had fissures with cordectomy survived without evidence of disease for periods varying from nine months to eleven years. Two with hemi-laryngectomies are also alive and well after one year, seven months and two years, four months. Eleven of the fifteen total laryngectomies have survived without recurrences for periods extending from eight months to two years.

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SUMMARY OF CASES

Case	Age	History & Symptoms	Examination	Pathology	Surgical Procedure	End Result
1.	F. R. 50	Hoarseness 3 months. No weight loss	Small ulcerating neoplasm, ant. third right cord; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 11 years
2.	M. G. 47	Hoarseness 5 months. No weight loss	Ulcerating neoplasm ant. and middle third left cord; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 10 years
3.	J. F. 61	Hoarseness 6 months. No weight loss	Ulcerating neoplasm ant. and middle third left cord and ventricular space; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 8 years
4.	J. K. 67	Hoarseness 9 months. Slight weight loss	Ulcerating neoplasm post. third left cord, ventricular space and ventricular band; slight fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 3 years. Death from pneumonia. No sign of malignant disease
5	M. G. 51	Hoarseness 7 months. No weight loss	Ulcerating neoplasm middle third left cord; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 7 years
6.	S. F. 49	Hoarseness 4 months. No weight loss	Ulcerating neoplasm ant. third right cord and ventricular space; no fixation	Intrinsic squamous cell carcinoma; anaplastic	Laryngofissure	Death from glandular and pulmonary metastasis, ninth month
7.	M. G. 37	Hoarseness 11 months. No weight loss	Ulcerating lesion right cord, middle third and ventricular space; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 6 years
8.	F. S. 52	Hoarseness 11 months. No weight loss	Ulcerating lesion left cord, ant. third; no fixation	Intrinsic squamous cell carcinoma	Laryngofissure	Living and well 5 years

9.	E. P. 57	Hoarseness 8 months. No weight loss	Small ulcerating lesion left vocal cord; no fixation	Intrinsic squamous cell carcinoma; non-keratinizing	Laryngofissure; postoperative irradiation	Living and well 18 months
10.	O. S. 54	Hoarseness 1 year. No weight loss	Large mass covering almost entire left cord, ventricular space and band, with subglottic extension	Intrinsic squamous cell carcinoma; keratinizing	Hemi-laryngectomy	Living and well 19 months
11.	G. G. 51	Hoarseness 15 months. Weight loss 10 lbs.	Ulceration at middle third right cord; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 13 months
12.	T. S. 64	Hoarseness 1 year. Weight loss 11 lbs.	Papillary lesion posterior and middle third, left cord; no fixation	Intrinsic squamous cell carcinoma; anaplastic	Laryngofissure	Death from pulmonary metastasis, 15 months
13.	R. B. 49	Hoarseness 14 months. No weight loss	Ulcerating neoplasm right cord, anterior third; no fixation	Intrinsic squamous carcinoma; keratinizing	Laryngofissure; postoperative irradiation	Living and well 10 months
14.	J. F. 60	Hoarseness 8 months. No weight loss	Ulcerating neoplasm left cord, ant. third ventricular space and band with subglottic extension	Intrinsic squamous cell carcinoma; keratinizing	Hemi-laryngectomy	Living and well 14 months
15.	S. F. 59	Hoarseness 2½ months. No weight loss	Ulcerating neoplasm anterior third right cord; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 9 months
16.	J. H. 67	Hoarseness 1 year. No weight loss	Ulcerating lesion right cord, post. third with subglottic extension; no fixation	Intrinsic squamous cell carcinoma; keratinizing	Laryngofissure	Living and well 14 months
17.	A. K. 58	Hoarseness 2 years. Weight loss	Ulcerating neoplasm rt. cord entire length, left ant. third rt. ventricular space and band involved; right fixation	Squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 2 years 5 months

SUMMARY OF CASES (Continued)

Case	Age	History & Symptoms	Examination	Pathology	Surgical Procedure	End Result
18.	K. G. 48	Hoarseness 9 months. Weight loss	Bulky ulcerating neoplasm, rt. cord, ant. third left, rt. ventricular space and band; right larynx fixed	Squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 7 years
19.	G. H. 51	Hoarseness 11 months. Weight loss	Bulky ulcerating ant. midline subglottic neoplasm, entire right cord involved; fixation, right	Squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 5 years. Death from angina
20.	B. J. 50	Hoarseness 13 months. Weight loss	Ulcerating neoplasm left cord, ventricular space and band; fixation	Squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 6 years
21.	J. F. 51	Hoarseness 7 months. No weight loss	Ulcerating neoplasm right cord, ventricular band and space, ant. third cord involved; fixation	Squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 21 months
22.	P. 53	Hoarseness several months. Weight loss	Bulky lesion left cord, ventricular band and space, rt. ant. third involved; thyroid gland invaded by extension	Squamous cell carcinoma; keratinizing	Total laryngectomy with thyroidectomy and neck dissection	Death 11th postoperative day from pneumonia
23.	F. N. 49	Hoarseness 7 months. No weight loss	Bulky neoplasm left cord, ventricular space and band; fixation	Squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 6 years
24.	R. B. 63	Hoarseness 7 months. No weight loss	Ulcerating neoplasm entire right cord, ventricular space and band, subglottic extension; right larynx fixed	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy; postoperative irradiation	Living and well 2 years 7 months

25.	E. T. 55	Hoarseness 11 months. No weight loss	Ulcerating neoplasm entire left cord, ventricular band and space, ant. third right cord; fixation, left	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy	Living and well 2 years 3 months
26.	R. W. 57	Hoarseness 9 months. No weight loss	Ulcerating neoplasm both cords; subglottic extension posteriorly; partial fixation, right	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 13 months
27.	H. C. 56	Hoarseness 14 months. No weight loss	Ulcerating neoplasm right cord, ventricular space and band, subglottic extension; fixation, right	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Living and well 8 months
28.	J. B. 65	Hoarseness 5 months. No weight loss	Ulcerating neoplasm left cord, ventricular space and band, right ant. cord; fixation, left	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy	Living and well 22 months
29.	H. H. 61	Hoarseness 4 months. No weight loss	Ulcerating neoplasm left and right cord ant. third; subglottic extension	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy; preoperative irradiation	Cervical and pulmonary metastasis, 2 months
30.	R. T. 59	Hoarseness 3 months. No weight loss	Ulcerating neoplasm right cord, ventricular space; subglottic extension; fixation, right	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy	Cervical metastasis, 3 months
31.	T. S. 49	Hoarseness 4 months. No weight loss	Ulcerating neoplasm right cord, ant. third and ventricular space; fixation, right	Intrinsic squamous cell carcinoma; keratinizing	Total laryngectomy	Cervical metastasis, 4 months

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XXIX

SOME PRACTICAL CONSIDERATIONS WITH REGARD TO HEARING TESTS*

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Modern exact methods of making examination of the hearing function are revealing a startling high incidence of hearing impairment.

It is especially among school children that such examinations have been conducted on a wide scale, and an average taken of reports turned in from all over the country, totaling over a million cases, indicates that six per cent have damaged hearing; that is to say, about one child in every seventeen examined.

The proportion is naturally greater in adults, because a considerable percentage of childhood defects will persist into adult life, and many additional ones will be acquired.

The hearing is of all the special senses the most vulnerable. It is in danger during every ordinary cold and it is likely to be seriously involved in certain infectious diseases.

It is peculiarly susceptible to various toxines originating within the body, and as has been emphasized of late, to many chemical agents introduced from without.

To these must be added the effects of excessive noise. Modern civilization has brought to us many highly prized comforts and luxuries but it has also brought to us a tremendous deal of din, for which our delicate ears must pay the price.

They cannot else but suffer from continuous exposure to the noises of traffic and transportation, and commerce and construction. Especially harmful, as experience proves, are the terrific grinding or pounding noises of some machine shops.

We are sure it is not too much to say, that with properly conducted hearing tests (that is, when they are made with instruments

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of precision in sound-proof rooms by capable examiners) at least ten per cent of the population as a whole will show at least a ten per cent loss of hearing. Ten per cent of the population of this country gives in round numbers a staggering total of 13,000,000 persons.

It is true that those who are at the lower limit with only a ten per cent loss are not seriously handicapped, but from there upward it becomes increasingly serious; so much so, that hundreds of thousands of children experience difficulties in acquiring an education, and hundreds of thousand of adults, difficulties in acquiring a gainful vocation.

According to the U. S. Bureau of Education there are in this country about 500,000 children who need special educational provision on account of defective hearing, compared to about 66,000 who need special education on account of defective sight.

A particular example to illustrate that the prevalence is not a matter of unconcern to the state, is to be found in army statistics. In the world war, 40,000 young men otherwise well and healthy were rejected for army duty because of deafness. The records further show that the same physical defect is one of the commonest causes assigned for discharge from the services. Such being the facts, does not the matter of deafness prevention seem to assume the proportion of a major national health problem?

Certainly the matter of hearing tests in its practical aspects seems to be a subject well worthy of the earnest consideration of medical men.

In all efforts to lessen the widespread evil of deafness, the first and foremost requisite is its detection—and otologists are everywhere agreed, that to be of most value, detections must occur at any early date—early in the life of the patient and early in the stage of the malady.

It is important to keep in mind that hearing tests are made for different purposes. The purposes may be briefly stated as follows: (1) In order to make a differential diagnosis in case of impaired hearing. (2) In order to determine the need of hearing aids, and if found needed, the type to be preferred. (3) For the examination of school children in order to evaluate their ability to follow instruction, and if a defect is discovered, as a help in determining the proper classification for education. (4) For examination of preschool children for the same reasons. (5) In order to determine fitness for occupations in which good hearing is essential. (6) To

determine fitness for military duty. (7) As a gauge of the efficacy of a particular method of treatment.

In addition, certain individuals may desire to have information of the state of their hearing function for aesthetic or social reasons. Not to be overlooked is its importance in certain cases in the consideration of marriage, because of the known influence of heredity.

These different purposes named naturally call for different testing procedures. This fact, I am sure, will be readily acknowledged by all who know anything at all about the subject but, unfortunately, it is not always strictly kept in mind and followed in practice. The result has led to some confusion, and has tended to bring a certain discredit upon hearing tests in general.

All hearing tests may be broadly divided into two classes, namely: (1) those made to simply gauge in a general way the capacity of the hearing function and (2) those made for the purpose of discovering the underlying nature of the trouble.

In the former case, we want merely to know the degree of loss. Tests of this kind are simple tests of hearing acuity. They can be made satisfactorily by laymen.

In the latter case, we want to know the kind of trouble that is causing the defect. The examination, therefore, has to be much more comprehensive, for its object is to make a diagnosis. Tests of this kind can of course only be made by otologists.

Obviously, diagnostic requirements include both kinds of tests. In the older textbooks they were generally referred to as "quantitative" and "qualitative" and they were carried out by separate procedures.

For the quantitative test we used the watch, the voice and the whisper; for the qualitative test we had the tuning fork, the monochord and the Galton whistle.

These old methods were of necessity crude and inexact. Hearing was estimated on the basis of the distance intervening between the subject and the sound. There was no guarantee of the volume of sound, and the intervening distance laid the procedure open to serious error because of surrounding noise.

Then came the electric audiometer, which overcomes these difficulties. It eliminates the uncertain factor of distance, and measures the hearing on the basis of sound intensity which can be varied at

will and determined at any point with mathematical precision. Furthermore, provision is made for a variation of the pitch of the sound over a range of six to eight octaves. Here then is an instrument which beautifully fulfills the two-fold requirement of the diagnostic test. It was in fact designed for this express purpose.

The electric audiometer was, it ought to be emphasized, invented by the otologists with the aid of the physicists for special otological needs. Theoretically it is an instrument which we should expect to find in every up-to-date otologist's office. But what has happened? Ear specialists as a whole show very little enthusiasm in regard to it, and only a relatively few employ it regularly in their work.

On the contrary non-medical persons are over-enthusiastic as to its merits and have in a measure monopolized its use for their particular purposes. It is especially in favor with various agents and firms interested in the sale of hearing aids, who take every occasion to exploit it for commercial reasons. They regularly use it in testing prospective purchasers, on the ground that they can thus diagnose their difficulties and exactly supply their needs.

There has of late sprung up in our very midst a new group who style themselves audiometrists, and who threaten to become a problem for otologic practice, closely akin to that which optometry has already become in the field of ophthalmology.

Newhart of Minneapolis, Jones of Los Angeles and others have called attention to this danger.

Jones intimates the fault is largely our own. We are making the same mistake which physicians made when they ignored the importance of manual manipulation and thus opened the door to the osteopathist and the chiropractor; or when they neglected in practice the known principles of neuro-psychiatry and made way for Christian Scientists and other drugless healers; or, as more recently, the ophthalmologists when they failed to make better practical application of their knowledge of physical optics, with the result that now, when it is too late to remedy the situation, 75 to 85 per cent of the refraction work in many communities goes to the optometrists.

Shall we profit by these examples? If not, we shall lose much, but the general public will lose more. We will lose clientele, but they will lose in frequent instances of poor and improper advice.

At present the activity of the audiometrists is supposed to be restricted to the problem of providing hearing aids, but depend upon it, it will become enlarged, and extend into fields where much harm will be done.

The public is not given to fine distinctions and it will inevitably tend to impute to them an understanding of otological problems of wider scope. Human nature being what it is, they will probably not be over-modest in accepting the imputation.

Not in this field alone, but in others, too, we may find non-medical use and abuse of the audiometer. It is too often improperly and unnecessarily used by nurses and teachers and other lay workers, as in the examination of school children, where only simple acuity tests are really required.

Complicated audiograms are often made without any understanding of their true implications. Most otologists are reserved in their interpretation of the audiogram. They realize that exceptions to the rule are common and that many factors must be considered before drawing conclusions. Not so with the non-medical individual. He rushes in where angels fear to tread and his opinions are always positive and unconditional.

Thus, we see that the audiometer is neglected in quarters where it should be used; and used or abused where it were better neglected. We see an instrument that was especially designed for diagnostic reasons mostly employed by non-medical persons and in fields where diagnosis is not or should not be an object.

There is little advantage in citing an evil unless some way can be found to correct it. Is there anything that can be done in this case?

The first thought is to secure legal restrictions. It is, however, very doubtful if this would be a wise course. Efforts of this kind usually bring unpleasant repercussions, and in the end serve no good purpose. The causes of the trouble seem to be within our own ranks. We must analyze the causes and remove them.

First of all, let us recognize the science of audiometry as a worthwhile, dignified branch of our specialty. It will well repay us to take sufficient time to fully master its principles and their significance in practice. We must in particular give greater attention to their application to the problem of hearing aids. We cannot as yet

prescribe hearing aids with the same precision that oculists can glasses; but we can even now on the basis of a thorough audiometric study give much helpful advice and prevent many ill-advised steps.

Remember as yet we hold the place of vantage. Most patients who need hearing aids come first to us. We must tell them what to do. Incidentally, we can throw the weight of our influence in the direction of those firms who do not overstep their proper bounds. There is another angle to the question which ought to be considered. Hearing defects, as pointed out, are amazingly common, and there are many persons who for one reason or another need to have their hearing tested.

School children have to be examined by the wholesale, and frequent tests have to be made to determine fitness of employees for their work. Generally no extended hearing examination is required so we cannot expect all these cases to be brought to the consulting room of the otologist, and we would not wish them to be. Provision, therefore, should be made for hearing tests by laymen, but the point is they should be simple acuity tests, and not attempts to duplicate the complicated procedures which otologists have to use for diagnostic work. The multitone or pitch audiometer is out of place.

It is a mistake to regard any one instrument, no matter how excellent, as applicable to all cases. It is the same sort of mistake that is made when a new and excellent remedy, such as sulphanilamid, is prescribed indiscriminately in all kinds of diseases.

When the object is merely to gauge hearing power and nothing more, a simplified instrument will answer the purpose. But it should be of the audiometer type, and just as truly an instrument of precision as that made for more difficult work; in other words, an audiometer minus the provision for pitch testing.

Hearing tests for school children is a subject that has of late received a vast deal of attention. For this problem a very special system has been worked out, which in spite of some defects has proved fairly satisfactory. A modified audiometer (known as the 4A) is used, which by means of a phonographic attachment, can reproduce a series of numbers with varying degree of loudness. The children are taken in groups and are instructed to write down on a prescribed blank form the numbers as they are spoken with a gradually diminishing intensity. The state of hearing is estimated according to the correctness of their answers.

It is almost equally important to make hearing tests of the preschool children, but for them no provision has been made. The 4A test, which is in effect a sort of written examination, is obviously unsuitable. Experience, in fact, shows that it is also not very well suited to school children of the primary grades. For these very young children your test must be very simple and of short duration, and one that does not exact any sustained attention.

Hearing tests are frequently required for occupational reasons. There are many industrial concerns which require routine examination of all employees, and this will include the hearing. Such tests for the most part are rather perfunctory and it would be absurd as well as impractical to attempt to use in all cases the pitch type of audiometer and to make audiogram charts. A simple acuity test is generally all that is required, though naturally for positions of a certain kind more thorough-going examinations will be needed.

The stenographers, for example, must have an acute hearing for speech.

The musician will want to have a perfect hearing for all musical tones, and will need a pitch audiometer test. Individuals whose duties require them to listen to certain high pitch signals will need to pass tests covering the high frequency sounds.

Aviators are expected to have a perfectly normal labyrinth and will have to undergo a thorough examination of the acoustic as well as the static function.

A good deal of emphasis is generally placed on the hearing faculty in connection with military duties. During peace times the Army or Navy surgeon may well indulge himself to the fullest extent—running the whole audiometric gamut, and finishing with a beautiful chart. But aural examination of recruits in war time is, as I know from experience, a different proposition. With only a minute or two allowed for each applicant as they file by from one room to the other, the test must be quick and snappy. If you stayed each case for an ideally perfect audiometric examination, the war might be over before they got to their posts.

It is evident that the kind of hearing test to be employed must be governed by the existing conditions. Time, convenience, and attendant circumstances will sometimes from a purely practical viewpoint have to be considered. It is generally a waste of time to attempt to get more information than is needed. One should limit himself to actual indications.

If the object of the test is to make a diagnosis, then of course a thorough-going test is indispensable, but as pointed out, for every case where diagnosis is the purpose, there must be a hundred cases in which a simple acuity test is all that is required.

I am convinced that there is a big field for a simple but accurate unitonal instrument, not only for the laymen, but for the doctor's use in circumstances where a complete multitontal testing procedure is not indicated. Preferably there should be an interrupted tone, like that of the watch tick, because a sound of this type is more readily perceived as it merges from silence into the lower threshold; also we are more likely to be aware of its disappearance as it fades out of sensation. We know how difficult it is in the case of a gradually decreasing humming sound to distinguish between a real fading sensation and a lingering memory image. The psychological basis is the same as that underlying the experience of a slight steady pressure against any part of the body. It may fail to come into consciousness, when we would be readily aware of a repeated tap of the same slight force.

Also in favor of the watch-tick type of sound is that it will not be confused with a possible tinnitus, something that can easily happen in the case of humming or buzzing sound. This advantage is of especial importance in testing persons who are not particularly keen in the appreciation of their own sensation. The watch tick is ideally adapted for testing very young children. Ordinary watches are of mixed tones without definite pitch. The watch-tick sound for testing should have a definite frequency that is best set at about the rate of 1000 cycles. The rationale is that this is the most important pitch level for perception of speech. The speech range extends from about 300 to 3000 cycles. The vowel sounds are in the lower levels while the consonants which are more important for speech appreciation are at the higher level. Good hearing at the 1000 cycle level is a practically safe guarantee of good hearing for speech.

Now, as regards the matter of precision, there need be no sacrifice on that score. In the first place, there should be an accurate calibration of intensities, just as for any other audiometer, and in the second place it is well to know that with regard to hearing tests, precision in results depends upon other factors besides the instrument.

We talk glibly about precision measurements of the hearing, as though all we needed was to have a finely made instrument, and

we could then measure the hearing as accurately as we could measure a piece of cloth.

In a recent excellent article on the subject of hearing tests, Jones points out the general neglect by otologists of the employment of instruments of precision compared to their general use by ophthalmologists. If the Man from Mars should visit our clinics, Jones writes, he would be amazed and mystified by this obvious inconsistency.

We were advised last summer that the Man of Mars had come to within some trifling thirty-six million miles of our planet, but for unknown reasons he never saw fit to step ashore, and from the present outlook, there is no immediate prospect of our getting across to him. Perhaps, therefore, for the present it would be just as well that we confine ourselves to considerations purely mundane; in other words, that we keep right down to earth.

This remark must not be taken in any sense as disapproval of precision methods, which on the contrary are heartily favored, but only that we are committed to a strictly practical discussion by the title of this paper.

What I wish to bring out is that we must not be led into supposing that a hearing test instrument of precision is any guarantee of precision of results. There are certain factors of personal as well as environmental origin which stand in the way. The chiefly environmental factor is the surrounding noise. Although from habit we have become in a great degree unconscious of the fact, we live for the most part, in an atmosphere of considerable noise.

If you happen to have a room looking out upon a busy city street and a window which can be easily open and closed at will, you can readily get some idea of street noise by the roar and rumble which will burst upon you when the window is opened, contrasted against the comparative quiet when closed. But the seeming indoor quiet is only relative. Scientific surveys show that there is a general condition of indoor noise even in residential districts of cities.

The noise level of the average city doctor's office has been found to range between 25 and 35 decibels. This equals about one-fourth of normal hearing, which means examinations made in such a noise could miss a hearing loss that is less than 25 per cent. This is of practical importance because it is the slight and beginning impairment for which the greatest good can be done.

There are some who maintain that since we habitually live in the midst of noise, it is all right to make your hearing test in a noisy environment. But how could we agree as to the proper level at which the test should be made? Different offices will differ in this respect, and the same office at different times of the day. Even while tests are being made there are likely to be wide fluctuations due to street traffic, etc., of as much as 20, 30 or even 40 decibels. It is only by having a sound-proof room can you avoid errors due to fluctuating noise, and get a true measurement based upon normal as a standard. A room perfectly noise free is difficult to attain, but without great expense you can insulate against disturbing noise and make it silent enough for practical purposes.

The other order of factors which interfere with precision measurements of hearing are of even greater influence, yet they are usually accorded comparatively little attention. I refer to the personal equation—not only on the part of the person being examined, but also on the part of the examiner.

While the subject is under examination, the elusive psychic element comes into play. Every physician of experience knows how uncertain are a patient's responses regarding the character and degree of his subjective sensation—even at the best. In the measurement of hearing we employ low or minimal threshold sensations, for the appreciation of which one needs to be keenly alert. If we have to deal with individuals who are stupid or inattentive the answers will be wholly unreliable. Naturally, in such case, the records of the examination must be unreliable in spite of the best instrument of precision. For, bear in mind, in every hearing test you are absolutely dependent upon the responses of the person being examined.

On the other hand, serious errors may arise which are attributable to the examiner. A proper examination of the hearing demands painstaking care, close observation and discernment. When made by persons who are lazy, careless or indifferent, the records may be worse than worthless. In the survey made of school children, using the same instrument and the same standard, the reports varied widely; some were as low as less than one per cent and some as high as over 12 per cent. We are convinced that these discrepancies are in great measure due to differences in the personal equation of the examiners.

When we have to entrust hearing examination to others, we must be sure that they are competent and reliable persons. They ought, if possible, be persons versed in the general principles of

psychophysics, or at least they should be drilled as to the precautions to be taken to avoid gross errors on this score.

The subject of hearing tests is one of growing interest and importance and it has been immensely advanced of late by some valuable contributions. In this paper, as I need not tell you, there is no pretense to anything like a comprehensive survey. The idea has been rather to consider the subject from a very realistic point of view, and in particular to point out certain trends that ought to be combatted, and some reforms of policy and practice that might be considered.

SUMMARY

Hearing tests of the diagnostic kind are obviously the province of the ear specialist, and it seems advisable that the instrument particularly designed for this purpose be kept in his hands. However, we should not fail to recognize that simple acuity hearing tests may very well be made by laymen, and considering the great number of such examinations that have to be made, this is the practical course.

Accordingly, we suggest an electrically operated unitonal instrument, of the watch-tick type as suitable for this purpose. We believe, for reasons stated, that the interrupted 1000 cycle tone will give a fairly representative estimate of conversation hearing. The intensity changes must be as accurately calibrated as in the best of audiometers. Practical requirement will be best served if the dial reading will be in percentages of normal.

After all, we have got to admit that absolute precision in hearing tests is an unattainable ideal in spite of instrumental precision. Noise is a disturbing factor and can be in a great measure eliminated. The personal factors, although they receive relatively little attention, are of much greater influence than usually realized. Since they are concerned with psychic phenomena they cannot be accurately calculated and controlled. However, it is believed a better knowledge of the principles involved and stricter observance of them in practice would materially help in cutting down the usual margin of error.

1606 TWENTIETH ST.

XXX

THE VENOUS CIRCULATION AS A FACTOR IN OSTEOMYELITIS OF THE SKULL*

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Osteomyelitis may originate in any bone of the head and extend to and involve any or all the rest of the head bones (except the mandible). Infection is transmitted from one part to another in various ways, the most important, perhaps, is along the venous spaces. This is particularly true for certain types of bacteria especially the staphylococcus which is the usual organism in osteomyelitis of the skull.

For purpose of description the venous system of the head may be divided into three parts: viz., intracranial, extracranial and the intercranial or diploic system.

The intracranial includes the veins of the brain and upper part of the spinal cord, the meningeal veins and the collecting spaces or dural sinuses which continue outside the skull as a part of the extracranial system, the rest of which comprise the venous network in the soft tissues outside the bony framework of the head. At certain points the two systems are joined by a connecting or emissary vein which usually anastomoses with the intercranial system and may serve as a channel by which infection might reach the diploe.

The intercranial or diploic veins sometimes referred to as the veins of Breschet are located in bony channels which run irregularly through the cancellous tissue between the external and internal tables of the skull. Their presence is an important factor in the extension of infection involving the marrow spaces of the calvarium, especially the rapid widespreading type. They possess certain peculiarities which favor bacterial invasion and the growth of infection.

On account of the facility with which the arterial blood reaches the venous spaces in the bones of the skull, these veins, in addition

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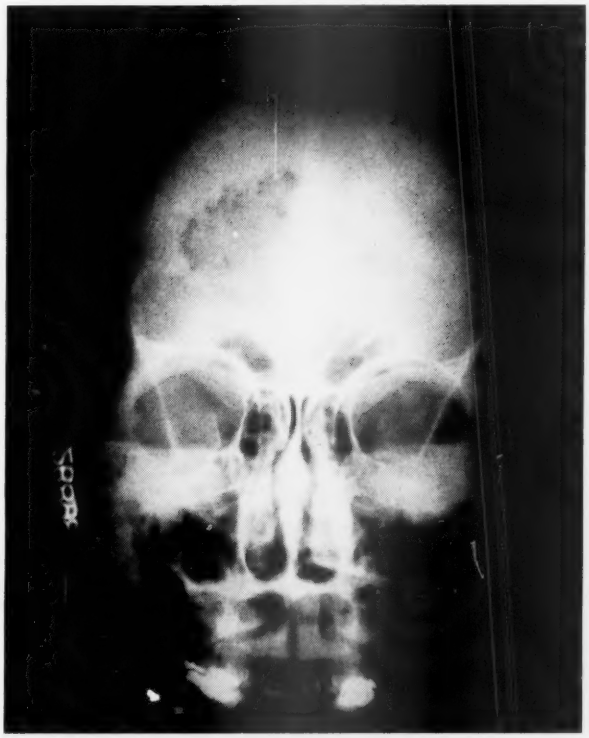

Fig. 1. X-ray of head showing congenital varicosed area in immediate relation to the right frontal sinus.

to the usual function of veins, must be considered as nutrient vessels. There are no corresponding arteries, the arterial supply consisting of many small vessels which enter the bone at different points and quickly break up in the diploic structure. These small arteries are especially numerous in the substance of muscular attachments.

The chief supply to the calvarium, however, is from the intracranial surface where many branches enter the bone along the course of the meningeal arteries. Attention has been called to the importance of this arrangement by Furstenberg,[1] who found collections of fluid between the dura and internal table in advance of the osteomyelitic process. The pressure of the fluid would tend to interrupt

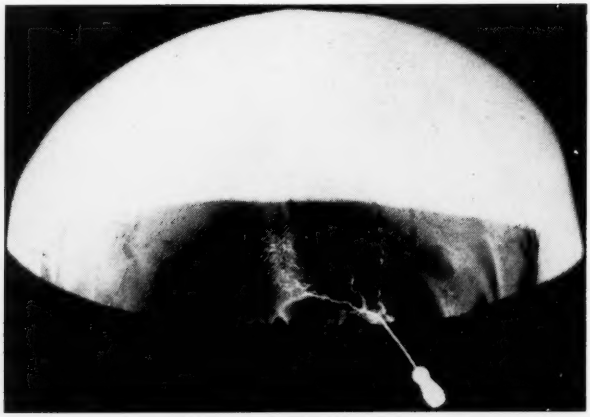

Fig. 2. X-ray of skull cap showing left frontal diploic vein injected with bismuth. Note its intimate relation to the upper angle of the frontal sinus, also connection with the superior sagittal sinus.

the blood supply thereby hastening the suppurative process in the bone.

The size of the diploic veins is variable and seems to have no relation to the thickness of the bone. While there may be little difference in the size of the two ends of a given vein, there are often greatly dilated areas along the course of the main channel, especially in the region of the frontal and parietal eminences. Many smaller channels enter these areas from either side, as well as branches which perforate the inner table to connect with the meningeal and cerebral veins, thus providing a direct route by which infection might reach these structures.

The diploic system of veins has no valves; the blood may flow in either direction depending upon the pressure within the veins. In fact there is a continuous oscillation of the intracranial and the extracranial venous pressure. It may thus be seen that the blood current is very sluggish at certain points which of course would favor bacterial invasion. Furthermore, if one end of a diploic vein be occluded by septic thrombosis the growth of the thrombus would be favored by a further slowing of the blood due to the obstruction. The presence of eddies in the current at certain points is also a factor which favors the pathologic process.

Fig. 3. X-ray of left side of skull cap showing the posterior temporal diploic vein injected with bismuth. The dark lines are grooves for the middle meningeal vessels. Note they cross at right angles.

The walls of these veins are very thin and imperfect, areas are often found where the vessel wall is hardly demonstrable. In fact when one injects such a vein the injection fluid is seen to spread out into the diploe instead of filling the smaller channels. Such points offer little resistance to the invading organism.

The diploic venous system arises from a vascular network on the interior of the skull which as development proceeds becomes included in the construction of the marrow spaces and is fairly complete by the fifth year. They converge to form four main channels on either side, the frontal, anterior and posterior temporal and the occipital. Any one or more may be absent on one or both sides; but when all are present they form a complete network of canals throughout the cranial vault. They branch freely crossing the sutures even in the midline which exposes all parts of the skull to possible attack by an osteomyelitic process. As a rule, however, there is a definite barrier to such a process at the sutures where the internal and external periosteum join. This is particularly true with the persistent suture or with those that normally fuse late in the development. These veins communicate above with the lateral lacunæ, superior sagittal sinus or with the extracranial circulation through the parietal emissary veins. The main channels pass downward to end in an emissary vein or break up into many small veins which perforate the external table to communicate with the veins outside the skull. Other branches pass inward to join one of the dural sinuses or meningeal veins. This arrangement is of great clinical importance as it is the chief port of entry for invading organisms.

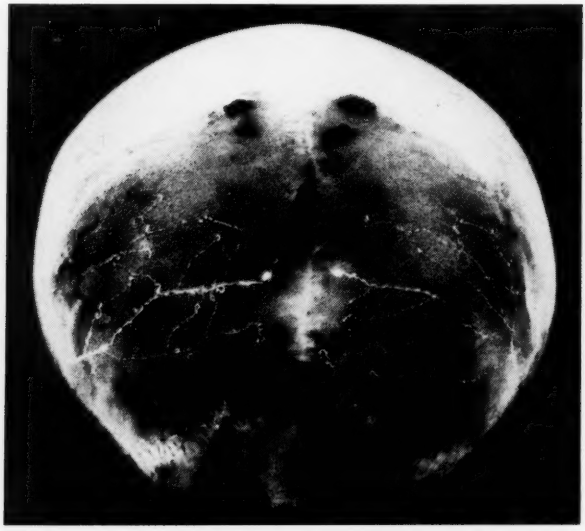

Fig. 4. X-ray of the back part of the skull showing the parietal bones and the upper part of the occipital bone. Bismuth is seen in the grooves for the meningeal veins which connect with the parietal emissary veins. The dark lines are channels for the occipital diploic veins. Note how they cross the lambdoid suture without interruption.

Early in development, each of the cranial bones has an independent vascular system including an emissary vein, but in the adult specimen there is a great variation in the arrangement of the diploe and venous channels. Many of the emissary veins become obliterated as the diploic structures of the different bones unite with the closure of the sutures. If the suture closes early in development, there is more likely to be a complete fusion of all the elements including the diploe and the diploic veins. Evidence of a suture may thus disappear completely as is ordinarily the case with the interfrontal suture, in which event, an infection in the bone would not be limited by the suture. On the other hand, if the suture remains open or the cancellus structure is not connected, the pathologic process would then probably stop at this point.

Of all the bones of the skull the temporal is most frequently attacked by an osteomyelitic process, yet seldom do we encounter an

extension from it to other bones. The reason is obvious, the sutures tend to remain open throughout life, thereby limiting osseous involvement to the individual bone. If extension to an adjoining bone does take place it will most likely be found to involve the sphenoid across the petrobasilar suture[2] or by way of the posterior temporal diploic vein through its connection with the mastoid emissary vein, involve the parietal and occipital bones. The suture has a tendency to close first at these two points.

As a factor in the origin and spread of osteomyelitis in the bones of the skull the frontal diploic veins are of first importance. These veins as well as the diploe are often in intimate relation to the frontal sinus, which develops by projecting itself upward into the vertical portion of the frontal bone between the external and internal tables, displacing the cancellous structure as it proceeds. The number and position of these veins will thus be found to vary with the degree of development of the frontal sinus. A layer of compact bone of varying thickness and density is laid down immediately beneath the lining mucosa which separates the air cell from the diploe and may constitute a decided barrier to the transmission of infection. When pneumatization of this portion of the frontal bone is complete, there remains no cancellous substance in the anterior and posterior sinus walls. If, however, development has been arrested at any point, diploic structure in varying amounts may then be present in the anterior and less often in the posterior wall.

A region of tremendous clinical importance because it can be demonstrated by the x-ray is the area immediately above the frontal sinus where the two walls approach each other. Here the diploe are frequently found to be in intimate contact with the air cell, a situation which might readily allow a suppurative process in the frontal sinus to pass directly into the diploe. When one studies a number of frontal sinuses in serial sagittal section, there is found a great variation in the construction of this area, as well as in the sinus walls, not only in different subjects but at different points in the same specimen. This fact then makes every case an individual problem and calls for careful studies of this area on lateral x-ray films in all instances of injury to the frontal region, frontal sinusitis and suspected osteomyelitis, as well as infections involving the soft parts about the orbit where the clinical picture is uncertain. When the roentgenogram in such a case shows the lining compact layer to be thick and dense with no adjacent diploic structure, the chances of bone disease or intracranial complications are slight, therefore, treatment should be conservative. If on the other hand, cancellous bone

Fig. 5. Photograph showing the upper limit of the frontal sinus perforated by foramens for the transmission of veins which flow directly into the diploe.

is present in close relation to the sinus, the prognosis is to be guarded and more radical measures instituted to prevent diploic involvement.

One should hesitate to enter the frontal sinus through the anterior wall in the presence of acute infection when the x-ray film shows cancellous structure present. There is considerable danger even in chronic infections, as shown by the number of instances cited in the literature where osteomyelitis followed surgical attack by this route. By such a procedure two things happen which favor osseous involvement; (1) the periosteum is stripped from the bone at the sight of the attachment of the frontalus muscle, this interrupts the blood supply to the bone and exposes the open venules to the infection; (2) the use of biting forceps and rasping instruments may force contaminated material into the marrow spaces beyond the barrier set up by nature. Therefore, when external drainage is indicated, the approach should be made through the sinus floor where these dangers are greatly minimized. However, there is some danger here if the opening is made anterior to a line extended upward from the posterior lacrimal crest, as this area often contains diploic structure which may extend across the suture into the frontal process of the maxilla, or even into the base of the nasal bones, thus making this the most vulnerable region, as the diploe will be found to extend

Fig. 6. Photograph of the interior of the calvarium with transmitted light, which shows deep grooving not only for the meningeal vessels but also the diploic veins. Note the extreme thinness of the bone at certain points.

backward between the two frontal sinuses, being continuous with those of the posterior sinus wall and the crista galli. The venous blood from this region would drain into the superior sagittal sinus at the foramen caecum, through which communication is established with the veins in the upper part of the nasal cavity and the anterior ethmoidal veins at the forward end of the cribriform plate. By this arrangement infection might readily enter the diploe of the frontal

bone in this area from a superficial infection about the inner canthus, upper part of the nasal cavity, or from ethmoid or frontal sinus suppuration. Fractures involving this region are particularly liable to be followed by such a complication.

Varicosed areas in the diploic system are often found to involve the internal table behind and above the frontal sinus which in the roentgen picture presents a worm-eaten appearance not unlike that of an osteomyelitic process for which it might easily be mistaken, as the following instance illustrates. Fig. 1. In 1922, Mrs. S., age 34, was confined to the hospital on account of sinusitis complicating influenza. High fever and severe pain in the right side of the head were the prominent symptoms. These, however, had disappeared and she was about to leave the hospital when the rhinologist ordered x-ray films of the sinuses which when developed caused considerable commotion and no little anxiety on the part of everyone concerned except the patient who was clinically well and refused to take any notice of the new findings. She has since had no trouble and films made in 1938 show no change in this area.

1725 STATE ST.

SUMMARY

1. On account of certain peculiarities the venous channels of the head seem to offer favorable conditions for the spread of infection.

2. Of all the head bones the temporal is probably most frequently the seat of osteomyelitis, yet seldom is there an extension to adjoining bones.

3. The frontal bone is second in frequency of attack and is most dangerous, because of the tendency to spread to neighboring bones.

4. In view of the variations in the construction and the importance of cancellus structure in close contact with air spaces, it is essential that careful x-ray studies be made in all cases of injury or infections involving these areas.

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Roentgen pictures made by Geyman and Gates, Cottage Hospital, Santa Barbara. Photographs prepared by A. B. Streedain, University of Southern California.

XXXI

RESULTS IN THE LABYRINTH FISTULIZATION OPERATION FOR CHRONIC PROGRESSIVE DEAFNESS: REPORT OF CASES*

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PHILADELPHIA

It is the common knowledge of all otologists that temporary improvement of the hearing in cases of otosclerosis and certain other types of chronic progressive deafness can be obtained by an operation, whereby a fistula is made into almost any part of the labyrinth. During the past twenty years considerable experimental and clinical work has been done, particularly by Holmgren,[1] Sourdille[2] and more recently by Lempert[3] seeking to establish a technic that would give a practical method of permanently improving such cases. In properly selected patients all of the earlier operators have had no trouble in obtaining an improvement of the hearing immediately on fistulizing the labyrinth, but in the great majority of instances this improvement has been maintained only a few days to several weeks because of the subsequent closure of the fistula by bone regeneration. Recently more lasting improvement of hearing has been obtained by Sourdille[2] according to his reports, and most encouraging reports have come from Lempert[3] of the prolonged improvement of cases operated upon according to his technic.

It will be conceded by all having any knowledge of this subject that in suitable cases of conductive deafness a temporary improvement of hearing to a practical degree can be obtained by fistulization of the labyrinth. Moreover, it has been established that with such improvement of hearing there is also a lessening of the tinnitus so often present in such cases. Permanent improvement of the hearing and tinnitus depends almost entirely on the retention of this fistula in the labyrinth. When bone regeneration closes this fenestrum the hearing either returns to its preoperative level or becomes worse, dependent on the technic of the operative procedure.

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In the operation of such cases a favorable result may be considered obtained if there is permanent improvement of hearing to a practical level, a lessening of the tinnitus or prevention of further progression of the deafness. While all the factors necessary for obtaining favorable results are probably not as yet known, at least some of them have been ascertained by experience and a discussion of these factors should be profitable.

The factors necessary for obtaining favorable results may be divided into three main headings: (A) Proper selection of the patient; (B) proper operative technique; and (C) proper postoperative care.

SELECTION OF THE PATIENT

Under the heading of proper selection of the patient should be considered those factors that appear to make the patient a suitable one for operation. These factors concern particularly (1) the degree of deafness, (2) the status of the bone conduction, (3) the adequacy of the vestibular function and (4) the state of the drum, middle ear and canal wall.

1. *Degree of Deafness:* The amount and type of deafness become important considerations in the selection of patients for the labyrinth fistulization operation. In order that the hearing may be improved to a practical level it is necessary that the initial deafness be not greater than 60 or 65 per cent (estimated by multiplying the average audiometer readings at 512, 1024 and 2048 frequencies by 0.8). However, if the other factors remain favorable a degree of deafness of 70 or 80 per cent need not contraindicate the operation, but an improvement of 30 to 35 decibels at the critical frequencies would still not bring the hearing to a practical level; although such improvement may be benefical to the patient, particularly if the tinnitus is lessened. The complete loss of hearing of the highest frequencies such as 5792, 8192 and those higher is not an unfavorable prognostic sign if the hearing of the speech frequencies remain reasonably good. It may be questioned if patients with less than 35 or 40 per cent loss of hearing should be operated upon because such patients still have some serviceable hearing which may be diminished if the operation is not completely successful. It seems perfectly justifiable to operate upon patients with this degree of hearing or even less than 30 per cent loss if it can be determined that the deafness is rapidly progressive, or is accompanied by disagreeable tinnitus, or in cases where the patient's business or pro-

fession is such that even the milder degrees of deafness become a handicap. In the consideration of patients for operation it must be remembered that the operative procedure seeks to lessen the tinnitus and prevent further progression of the deafness, as well as to improve the hearing.

2. *Status of the Bone Conduction:* From the experience gained in the observation of cases operated upon it appears of extreme importance that the bone conduction remain good if good results are to be expected. The factors concerned in bone conduction are intricate and incompletely understood so that it is difficult to give any definite standards that will determine whether a particular patient is or is not suitable for operation as far as this factor is concerned. If a bone conduction audiometer receiver is available with which a standard pressure can be maintained against the bone to be tested and a suitable masker used, then this method becomes of value in the estimation of the bone conduction and definite figures can be obtained for each frequency.

The determination of the bone conduction by means of the tuning forks is a simple but apparently effective way of estimating the suitability of the patient for operation as far as this factor is concerned. When the tuning forks show a lengthened bone conduction, a markedly negative Rinne test and a Weber test giving lateralization to the deafer ear it is good evidence that this factor of bone conduction is favorable. When the bone conduction is found to be less than the air conduction with lateralization to the better ear, such a case should, as a rule be considered unfavorable for the fistulization operation; although it is quite possible that such a patient may still be improved by operation, because the estimation of the bone conduction may have been at fault due to unknown factors that are known to interfere at times with conduction of sound by bone.

3. *Adequacy of the Vestibular Function:* Normally functioning semicircular canals appear to be an important prerequisite for good results. It is quite possible for the vestibular mechanism to be dead and the cochlear function remain unaffected. In such cases on fistulizing the labyrinth the dizziness, nausea and nystagmus observed when an actively functioning labyrinth is fistulized, will be absent. This was exemplified in one of my cases (case No. 7) reported a few months ago. On opening the external semicircular canal no dizziness or nausea resulted and even after the membranous

canal had been opened no dizziness or nystagmus followed. In this case it was also observed that there was not the immediate improvement of hearing when the membranous labyrinth was exposed as is expected in the suitable cases. Similar experiences have not been reported and no definite statement regarding this point should be made from the observation of one case, but it seems logical and probable that improvement of hearing will not be obtained by the fistulization of a dead labyrinth. It is important, therefore, before considering operation on a patient, to test the vestibular function by means of the Barany and caloric tests. I believe those patients with weakened or absent vestibular function should be considered as unsuitable for operation.

4. *State of the Canal Wall, Drum and Middle Ear:* A healthy condition of the skin surface lining the canal wall, particularly the upper and posterior surfaces, seems essential. Any eczema or dermatitis of this area may cause postoperative inflammation or irritation which will impair the results or may possibly set up a labyrinthitis. An exostosis of the deeper canal wall may make it difficult or impossible to satisfactorily dissect free the cutaneous lining of the bony canal wall without lacerating it. It has been supposed that a perfectly normal tympanic membrane without perforations is essential for good results. It is quite likely that such a condition is necessary for the best results, but a drum that is thickened, retracted and scarred need not contraindicate operation if the other factors remain suitable. Three of my patients in whom at least fair results were obtained have had thickened and scarred drums and the history of having had suppurative otitis media at one time. It is also questionable if a patient with a drum having a perforation is not still suitable for operation.

The state of the middle ear may have some bearing on the results to be obtained by operation. It is supposed that the best results will be obtained in those typical cases of otosclerosis in which the drum is entirely normal and there is fixation of the stapes. It seems very likely true that the best results will be obtained in such cases, but I believe further experience will indicate that at least good results may be expected when the drum is not entirely normal and when there is not marked fixation of the stapes. The presence of an acute severe suppurative otitis media at one time may have caused some destruction of middle ear structures, fibrosis and adhesions, which may interfere to some extent with good results of the operation. More experience is necessary to determine if and how much such factors influence the results in the fistulization operation.

OPERATIVE TECHNIC

While the proper selection of the patient is of prime importance, the results of the operation would be poor or nil without close adherence to an exacting operative technic. The basis of this proper technic is an anatomical knowledge of the parts involved, skill and care in the handling of most delicate structures, patience and physical endurance. To obtain results according to Lempert's technic these requirements are strictly necessary.

There are certain particular points of this technic that are of special importance and should be given emphasis. From the beginning of the operation and throughout its course the greatest possible care must be exercised for the preservation of the cutaneous membrane lining the posterior and superior walls of the external auditory canal. In the removal of the bony canal wall and sulcus tympanicus, this membrane will be lacerated and destroyed unless extreme care is taken. Another delicate part of the technic is the exposure of the malleus. In this procedure it is easily possible to dislocate the incus requiring its removal, or to lacerate the tympanic membrane. The removal of the sulcus tympanicus, particularly in its upper portion, is an intricate part of the technic and requires great care and patience to avoid lacerating the drum. Finally the success of the operation depends on the proper technic in creating the fistula in the external semicircular canal and the fitting of the tympanomeatal cutaneous membrane over this fistula. Incomplete exposure of the membranous labyrinth may result in prompt closure of the fistula by bone regeneration, while too much exposure may result in a perforation of the membranous labyrinth. The success of the operation depends almost entirely on the proper handling of this part of the operation; although the previous steps are of importance, if the greatest possible amount of hearing is to be gained. When the fistula is completed there is an immediate improvement in hearing, but for this improvement to be maintained at its highest level it is necessary that the previous stages of the operation be completed accurately. For instance, if the tympanomeatal cutaneous membrane be lacerated, there will be no membrane available to cover the fistula, unless a new flap is dissected from the lower or anterior canal wall, and then it will have no connection with the drum. If the drum itself is lacerated the healing process may cause a retraction that will impair its efficiency. If the incus is dislocated the ossicular chain is broken and any sound impulses that may be carried through that channel are lost.

When such accidents occur, however, the operation need not be considered a complete failure, for improvement of hearing will still be present if the fistula remains open. This is illustrated in my first case operated upon 15½ months ago. During the early stages of the operation the cutaneous lining of the external auditory canal was badly lacerated so that it was impossible to use it to cover the fistula in the external semicircular canal. A skin flap was therefore dissected from the anterior canal wall and stretched across the canal lumen and placed so as to cover the incus, the posterior edge of the tympanic membrane and the fistula of the external semicircular canal. In the healing process this resulted in narrowing the canal lumen to about one-half its normal size with a band of tissue stretched across the lumen from the anterior to the posterior walls, with the drum visible below this band and the area of the antrum visible above it. I consider that in this patient I obtained one of my best results, as an audiogram on August 29, 1939, fourteen months after operation, shows an improvement of hearing from the preoperative level of 20 decibels for the 512 frequency, 30 decibels for the 1024 frequency, 35 decibels for the 1448 frequency and 25 decibels for the 2048 frequency, or an improvement of 21.5 per cent. The fistula remains patent.

In another patient both the cutaneous lining of the canal wall and the drum were lacerated, but the remnants of the membrane were approximated as closely as possible and a section of the torn cutaneous membrane placed carefully over the attic cavity, the incus and the fistulized external semicircular canal. These torn skin sections were kept in place by small pieces of gauze mesh. This patient's ear healed so well that the drum barely shows a scar of the laceration and the skin has completely lined the excavated mastoid cavity and antrum. An audiogram on August 25, 1939, seven and one-half months after operation, showed an improvement of hearing over the preoperative level of 15 decibels for the 512 frequency, 30 decibels for the 1024 frequency, 35 decibels for the 1448 frequency and 35 decibels for the 2048 frequency, or an improvement of 20.6 per cent. It seems quite probable that greater improvement in the hearing of these two patients may have been obtained if these accidents in the operative technic had not occurred, but they illustrate that such an apparently serious fault may still be consistent with at least a fairly good result.

It has been asked in what way the removal of the head of the malleus contributes to the results obtained. The reason for its removal appears to be that greater mobility of the drum is gained and

makes it possible to stretch the tympanomeatal cutaneous membrane to such an extent that the thinnest part of this skin membrane and probably frequently Shrapnell's membrane itself can be placed directly over the fistulized semicircular canal. It also apparently facilitates the sealing off of the epitympanic space. Whether or not the removal of the malleus head is necessary for good results in all cases is a matter for further experimental study. In three of my more recent cases I have not removed the head of the malleus and the immediate results have been good. It is too soon after operation, however, to be able to form any definite opinion on this particular point, but it is a matter I shall discuss later when time will have permitted further observation and study.

In general, as regards the value of the operative technic, it can be said that good results depend very largely on the proper method of fistulizing the labyrinth and that the other details of the operative procedure will probably add to the results obtained in proportion to the accuracy and completeness of the various steps. However, it can also be said that minor accidents in the technic may still allow of an improvement of hearing that may be considered good.

POSTOPERATIVE CARE

This is the least important of the factors that may influence the result of the fistulization operation, although carelessness or negligence in the postoperative care may lead to failure in an otherwise successful case. The chief things to consider under this heading are (1) maintenance of asepsis, (2) treatment of excessive granulations and (3) general care and advice.

1. *Maintenance of Asepsis:* It is just as important to prevent bacterial contamination of the operative site after operation as it is to insure an aseptic field before operation. This may not be easy to do as the ear requires frequent cleansing and dressing. Furthermore, carelessness on the part of the patient may allow the ear to become contaminated. Following the operation there is considerable thick exudative moisture in the operated area and covering the canal walls and drum. It is advisable that this exudate be allowed to drain away and therefore it is necessary that the patient change the sterile cotton or gauze dressing over the ear frequently, often two or three times a day. The patient must be warned to use care in doing this and to use only sterile materials. One of my patients developed a middle ear abscess, in the operated ear, six weeks after operation. The drum perforated and drained pus for about a month. This caused an im-

mediate decrease in the hearing but no systemic effects and no symptoms of labyrinthine stimulation. Before the abscess developed the hearing had improved 10 decibels for the 512 frequency, 30 decibels for the 1024 frequency and 30 decibels for the 2048 frequency. During the course of the ear infection the hearing was reduced from this level and for the past five months has remained at practically the same level, the last recording on September 1, 1939, showing 38.7 per cent loss, almost exactly the reading before operation. As the fistula has remained open as indicated by an active fistula reaction, I have attributed the reduction of the hearing to changes set up by the ear infection.

2. *Treatment of Granulations:* Exuberant granulations in the operative site are common and require burning with trichloracetic acid (25 per cent) or strong silver nitrate solution. Application of such agents probably also stimulate epidermization of the cavity. There will be an exudative discharge and formation of granulations for two to five months after operation. This necessitates frequent attention by the surgeon as well as care by the patient during this time.

3. *General Care and Advice:* After the patient is discharged from the hospital usually the postoperative dizziness has subsided sufficiently as to require no particular care. At times, however, the dizziness will persist for several weeks, particularly on stooping or turning movements. The patient must be cautioned against overstimulation of the labyrinth by exertion. On the other hand, he should be encouraged to maintain some activity in order to accustom himself to the changes in the equilibrium mechanism. The patient is apt to be apprehensive of this frequent vertigo, but apparently there is gradual adjustment of this reaction, and later it does not interfere with normal activity; although quick turning, cold air in the ear, blowing of the nose, etc., cause more dizziness than had been present before operation.

The patient may require some treatment and advice during the convalescence because of the changeable character of the hearing. After the gauze packing is removed from the ear at the end of seven or eight days, considerable improvement of hearing is usually noticeable. The hearing may be greater at this time than later when the tissues have healed, but at times the trauma to the tympanic membrane and other tissues may cause such a swelling and edema with perhaps some hemorrhage into the middle ear cavity that improvement of hearing is delayed until this reaction has subsided. Further-

more, a serous labyrinthitis may have developed from manipulation of the membranous labyrinth and this will delay improvement of the hearing, as well as increase the vertigo until this labyrinthitis subsides. During the weeks following operation the hearing will vary somewhat according to the amount of moisture present and how well it is drained off. Frequently there is considerable thick exudate that covers the drum and fistula area preventing the transmission of sound impulses. Frequent removal of the secretions is advisable to prevent this. If the patient develops a nasal or sinus infection during the convalescence, it is apt to reduce the hearing due to inflammatory reaction in the eustachian tube. This is similar to the impaired hearing accompanying many colds in anyone and the hearing improves again as the nasal and tubal inflammation subside. I have noticed this on several patients, and it is cause for apprehension on their part as to the effects on their hearing. These patients are for the most part of a nervous and emotional temperament and require careful handling and frequent encouragement.

The factors which have been discussed have a variable effect on the results obtained with the labyrinth fistulization operation. It is again emphasized that accidents during the operative technic may perhaps lessen the final result obtained, but may not necessarily cause a failure in the particular case operated. Permanent improvement of the hearing depends on the maintenance of the fistula in the labyrinth, and on the accuracy with which the other details of the operation are conducted depends to a large extent the amount of hearing that will be recovered.

REPORT OF CASES

At this point I should like to give a brief additional report on those cases which were reported seven months ago and which will be published within a short time. It is still a questionable point how long a time must elapse before it can be said that the danger of closure of the fistula by bone regeneration is past. It is only by close follow-up observation of operated cases that this point can be settled. The following is a brief outline of the progress of the eight cases reported seven months ago which were operated according to the technic of Lempert.

CASE 1.—E. H., a man aged 35, was operated on June 29, 1938, on the right ear. A preoperative audiogram showed (by air conduction) a 54.7 per cent loss of hearing in the right ear and 42.7 per cent loss in the left ear. Six months after operation an audiogram

showed 35.5 per cent loss in the right ear, two months later 34.4 per cent loss, and on July 29, 1939, 33.3 per cent loss.

At the end of 14 months following operation, therefore, the improvement of hearing is 21.4 per cent greater than before operation (a gain of 20 decibels for the 512 frequency, 35 decibels for the 1024 frequency, 30 decibels for the 1448 frequency and 25 decibels for the 2048 frequency). Audiograms of the unoperated ear during this time have shown a slight increase of the deafness. The fistula 14 months after the operation is very active.

CASE 2.—S. H., a man, aged 43, had the fistulization operation on July 13, 1938. An audiogram on July 12, 1938, showed 40 per cent loss of hearing in the right ear and 38.1 per cent loss in the left. His bone conduction was not good and he was operated because the tinnitus was very distressing and it was thought there was some possibility of improving it. The operation was a failure, as the membranous labyrinth was perforated, resulting in the loss of the vestibular function. The hearing after operation was considerably reduced and has not improved to date and the tinnitus has not become less. There is, however, some hearing still present in the operated ear and the bone conduction has not changed.

CASE 3.—C. P., a girl, aged 19, was operated on July 20, 1938. There was immediate improvement of the hearing, but it began to fade soon and at the end of three weeks from the operation, the hearing had diminished below what it was before the operation, and the fistula test was negative. On December 8, 1938, the external semicircular canal was again fistulized with again good immediate results. Within a few weeks, however, the hearing began to diminish with lessening fistula reaction, and on August 29, 1939, an audiogram showed 57.3 per cent loss of hearing, a decrease of 16.5 per cent from the preoperative hearing. There is now no fistula reaction present and the tinnitus which had been improved at first is present as before.

CASE 4.—E. W., a girl, aged 21, received operation on the right ear on August 29, 1938. Preoperative audiograms showed a hearing loss of 35.5 per cent in the right ear and 27.7 per cent in the left ear. Immediately following the operation the hearing was much improved, but within two weeks it had begun to diminish and at the end of one month from the operation the hearing was slightly worse than before operation, with return of the tinnitus. Her laby-

rinth was refistulized on January 5, 1939, with again improvement of hearing. During the subsequent few months audiograms showed a maintenance of improvement but the improvement has not been great. On August 29, 1939, just one year after operation, there was 30.7 per cent loss of hearing, an improvement of 5 per cent over the preoperative hearing. The fistula reaction is still present, but not as active as during the first month following operation. There has been no change in it, however, during the past three months and no change in the hearing.

CASE 5.—N. G., a woman of 44, had the right external semicircular canal fistulized on September 30, 1938. She had had a successful radical mastoid operation performed on the ear some years before. The present operation consisted in elevating the thin skin overlying the antrum and aditus region, fistulizing the external semicircular canal and replacing the skin over the fistula. A preoperative audiogram showed a hearing loss of 25.9 per cent. During the four weeks following the operation the hearing was worse than before operation, coincident with severe dizziness, nausea and vomiting which was attributed to a serous labyrinthitis set up by the operation. As this subsided during the second month the hearing improved but did not return quite to the preoperative level. An audiogram on August 22, 1939 (about eleven months after operation) showed a hearing loss of 34.7 per cent, a reduction of 9 per cent over the preoperative level. The fistula reaction remained very active for the three months after operation, but during the next month became gradually less active. For the past five months there has been no change in its reaction, but it is not nearly as active as during the earlier months. A peculiar feature of the case is the fact that at times there is considerable improvement of the hearing for periods of a few minutes to a day or two without any apparent reason.

CASE 6.—N. C., a woman of 38, had the fistulization operation on October 12, 1938. Preoperative audiograms showed a loss of hearing in the right ear of 61.6 per cent and in the left ear of 48.8 per cent. She gained considerable immediate improvement but this became worse during the first three weeks associated with severe dizziness and nausea. When this subsided there was marked improvement, and at the end of a month the hearing was better by 30 per cent. Three months after operation the hearing was improved by 25.3 per cent and the last audiogram on September 18, 1939, showed 41.3 per cent loss or an improvement of 20.3 per cent over the preoperative level (a gain of 25 decibels for the 512 frequency,

30 decibels for the 1024 frequency, 20 decibels for the 1448 frequency and 20 decibels for the 2048 frequency). It has now been almost exactly a year since the operation and the fistula test gives an active response. The hearing is not as good as at the end of the first month following operation, but the practical improvement is great and has been extremely pleasing to the patient.

CASE 7.—L. H., a boy, aged 14, received his operation on the left ear on October 19, 1938. Preoperative audiograms showed 46.9 per cent loss of hearing in the right ear and 53.3 per cent loss in the left ear. There had been a history of ear abscesses earlier in life and the drums showed thickening, retraction and scarring. The bone conduction was somewhat decreased with lateralization of tone to the better ear. When the external semicircular canal was fistulized there was not the usual dizziness and improvement of hearing noticeable in the other cases. During the weeks following operation audiograms showed about 20 per cent loss of hearing and to date it has not improved. The patient is unaware, however, of the decrease of hearing, because his preoperative hearing was so little that no difference is noticeable to him.

This case illustrates well the unsuitability of the patient for operation because of the decreased bone conduction and dead labyrinthine function.

CASE 8.—F. B., a woman of 40, was operated on January 11, 1939. Preoperative audiograms showed a hearing loss in the right ear of 61.9 per cent and in the left ear of 40.3 per cent. At the end of six weeks following operation the hearing of the operated ear (right) had improved to 25.3 per cent loss, an improvement of 36.6 per cent (a gain of 40 decibels for the 512 frequency, 40 decibels for the 1024 frequency, 60 decibels for the 1448 frequency and 45 decibels for the 2048 frequency). During the next five months there was a gradual loss of the improvement until on July 28, 1939 (6½ months after operation) the hearing loss was 40 per cent, an improvement of 21.9 per cent over the preoperative level. Although the greatest improvement of hearing gained by this operation has not been maintained, there is still considerable improvement which has been extremely beneficial to the patient. The fistula at present is active but not as active as during the first two months after operation. It must be assumed that there has been some regeneration of bone which has partly closed the fistula. The question is whether or not this regenerative action is still going on. The fact that there has been no hearing loss in the last two months is encouraging.

In a short time, I shall make a preliminary report of twelve additional cases operated within the last few months. The early results in most of these new cases have been gratifying but it is too early to be sure that the improvement of hearing thus far obtained will remain.

An analysis of the eight cases described above will reveal results of a variable character. Three of the cases have gained improvement of hearing, the first having retained an improvement of 21.4 per cent after 14 months, the second, an improvement of 20.3 per cent one year after operation, and the third, an improvement of 21.9 per cent eight months after operation. Two cases showed prompt closure of the fistula by bony regeneration. On reopening these external semicircular canals one of these cases has again shown closure of the fistula with a loss of 16.5 per cent over the preoperative hearing. The second of the reoperated cases has retained a fairly active fistula and an improvement of 5 per cent in the hearing at the end of nine months from operation. In two of the cases the hearing has been made worse, due to their being unsuitable for the operation, both having had decreased bone conduction and one having had a dead labyrinth. The remaining case, the one in which fistulization was attempted in a previously radically operated mastoid, has also shown a slight loss of hearing over the preoperative level.

The results obtained in these few cases emphasize the importance of those factors that have an influence on the success of the operation. The proper selection of the patient remains the prime prerequisite while the proper operative technic and postoperative care are of somewhat less importance, but still extremely necessary, if the greatest amount of hearing is to be gained. Three successful cases out of eight will not be considered as good results, but if it can be shown that these cases have gained a practical improvement of hearing, or a reduction of the tinnitus and have retained the fistula beyond the time that bony regeneration should have occurred, then the operation can be considered as demonstrating the possibility of permanently improving the hearing. As the standards for selection of the patient become understood and the operative technic be developed and improved, it seems reasonable to assume that a high percentage of patients will obtain improvement of the hearing by such surgical procedures.

2035 DELANCEY ST.

CASE 1. (E. H.) OPERATION 6-29-38

RIGHT EAR

Audiometer Frequencies	6-18-38	7-9-38	12-1-38	2-8-39	4-17-39	8-29-39
128	58	29	39	27	35	35
256	59	37	47	38	42	45
512	67	47	37	34	40	45
1024	53	42	37	36	35	20
1448	57	58	35	37	35	25
2048	85	73	59	59	60	60
2900	88	83	73	69	65	80
4096	88	83	76	84	85	85
5792	100	100	100	100	100	100
8192	100	100	100	100	100	100
Hearing Loss for Speech in Percentage	54.7	42.7	35.5	34.4	36.	33.3

LEFT EAR

Audiometer Frequencies	6-18-38	7-9-38	12-1-38	2-8-39	4-17-39	8-29-39
128	42	50	45	48	52	50
256	43	44	40	40	45	40
512	55	49	41	39	40	50
1024	38	42	49	46	45	35
1448	48	58	60	63	60	55
2048	67	73	77	81	80	80
2900	18	83	90	88	90	90
4096	82	82	84	82	90	85
5792	100	100	63	70	70	100
8192	100	100	60	64	70	100
Hearing Loss for Speech in Percentage	42.7	43.7	44.8	43.3	44.	44.

CASE 3. (C. P.) OPERATIONS 7-20-38 and 12-8-38

RIGHT EAR

Audiom. Freq.	7-1-38	11-8-38	12-17-38	1-10-39	2-7-39	3-14-39	7-28-39	8-29-39
128	34	54	22	15	55	65	45	65
256	41	62	35	36	55	75	65	75
512	51	66	52	60	65	70	65	75
1024	53	77	42	52	55	65	75	70
1448	53	70	47	47	65	75	65	70
2048	63	69	49	54	65	70	70	70
2900	55	79	63	52	70	70	75	80
4096	78	100	100	75	100	100	75	100
5792	100	100	100	100	100	100	100	100
8192	100	100	100	100	100	100	100	100
Hearing Loss for Speech in Percentage	44.8	56.3	37.9	44.3	50.4	54.7	56.	57.3

LEFT EAR

Audiom Freq.	7-1-38	11-8-38	12-17-38	1-10-39	2-7-39	3-14-39	7-28 39	8-29-39
128	35	55	53	57	50	30	30	50
256	45	47	43	45	40	45	50	50
512	57	51	59	68	60	50	50	50
1024	43	47	41	38	40	35	50	50
1448	45	47	46	57	55	45	40	45
2048	49	51	49	47	50	55	45	55
2900	55	52	54	63	60	55	55	60
4096	81	56	69	72	75	70	80	100
5792	100	100	100	100	100	100	100	100
8192	100	100	100	100	100	100	100	100
Hearing Loss for Speech in Percentage	39.7	39.7	39.7	40.8	40.3	37.3	38.7	41.3

CASE 6. (N. C.) OPERATION 10-12-38

RIGHT EAR

Audiometer Frequencies	9-26-38	10-22-38	11-5-38	12-5-38	12-12-38	9-18-39
128	65	58	43	47	52	65
256	70	48	70	54	54	70
512	85	47	45	48	57	60
1024	80	37	48	48	42	50
1448	80	37	43	41	42	60
2048	65	35	46	42	42	45
2900	65	57	53	35	32	50
4096	77	72	74	53	55	70
5792	100	100	100	100	100	60
8192	100	100	100	100	100	100
Hearing Loss for Speech in Percentage	61.6	31.7	37.1	35.7	37.3	41.3

LEFT EAR

Audiometer Frequencies	9-26-38	10-22-38	11-5-38	12-5-38	12-12-38	9-18-39
128	77	68	73	70	68	65
256	73	69	75	70	66	70
512	76	82	77	70	70	65
1024	67	70	64	63	68	65
1448	68	70	68	63	63	70
2048	39	45	55	38	48	45
2900	46	43	47	43	48	45
4096	67	75	68	77	73	70
5792	68	65	100	67	100	60
8192	100	100	100	100	100	100
Hearing Loss for Speech in Percentage	48.8	52.5	52.	45.6	49.9	46.7

CASE 7. (H. L.) OPERATION 10-19-38

RIGHT EAR

Audiometer Frequencies	5-11-38	9-24-38	12-10-38	1-21-39	9-27-39
128	33	41	42	43	35
256	38	43	48	43	40
512	48	53	45	46	45
1024	58	62	63	62	60
1448	55	54	55	53	55
2048	56	60	58	51	55
2900	56	62	58	56	55
4096	62	67	73	55	55
5792	100	100	100	100	100
8192	100	100	100	100	100
Hearing Loss for Speech in Percentage	43.2	46.9	43.3	42.4	42.7

LEFT EAR

Audiometer Frequencies	5-11-38	9-24-38	12-10-38	1-21-39	9-27-39
128	68	48	100	100	60
256	65	47	100	100	85
512	62	53	97	100	95
1024	67	62	95	96	90
1448	58	52	91	90	90
2048	76	84	100	90	100
2900	80	86	94	100	100
4096	86	90	100	100	100
5792	100	100	100	100	100
8192	100	100	100	100	100
Hearing Loss	54.4	53.3	77.3	76.	76.

CASE 8. (F. B.) OPERATION 1-11-39

RIGHT EAR

Audiometer Frequencies	11-19-38	1-5-39	2-6-39	2-11-39	2-23-39	3-9-39	4-28-39	6-16-39	7-28-39	8-25-39	9-28-39
128	64	56	19	27	15	15	20	30	40	40	45
256	59	55	24	23	20	15	20	35	45	50	50
512	72	74	47	32	32	35	45	50	55	60	60
1024	72	68	32	32	25	25	25	35	40	40	45
1448	87	85	42	41	35	40	35	45	50	50	50
2048	89	90	44	41	42	45	50	55	55	55	50
2900	85	90	69	68	72	65	45	55	60	65	65
4096	100	100	100	100	85	100	75	100	100	90	100
5792	100	100	100	100	100	100	100	100	100	100	100
8192	100	100	100	100	100	100	100	100	100	100	100
Hearing Loss for Speech in Percentage	62.4	61.9	32.8	28.	25.3	28.	32.	37.3	40.	41.3	41.3

LEFT EAR

Audiometer Frequencies	11-19-39	1-5-39	2-6-39	2-11-39	3-9-39	4-28-39	6-16-39	7-28-39	8-25-39	8-28-39
128	37	26	43	41	60	45	35	35	30	30
256	37	33	40	46	55	45	35	35	35	35
512	37	41	37	41	50	50	40	40	45	40
1024	47	46	48	51	55	45	45	55	50	50
1448	53	55	57	52	55	55	60	55	55	55
2048	64	63	65	60	70	65	65	70	70	70
2900	63	57	55	59	65	60	55	65	65	60
4096	74	78	79	73	80	75	85	100	100	85
5792	100	100	100	100	100	100	100	100	100	100
8192	100	100	100	100	100	100	100	100	100	100
Hearing Loss for Speech in Percentage	40.	40.3	40.	40.5	46.7	42.	40	45.3	44.	41.3

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XXXII

DIAGNOSIS AND TREATMENT OF CHRONIC DISEASE OF THE PARANASAL SINUSES*

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The fundamental requirement in the treatment of chronic sinusitis is a differential diagnosis exact enough to prevent the application of formidable surgical procedures to conditions for which they cannot logically be expected to produce any beneficial effect. It is lack of attention to this important detail that has had the effect of bringing surgical procedures, directed against chronic suppuration in the paranasal sinuses, into disrepute among the uninformed physician and layman, not, however, without justification.

Attempts at a solution of the problem of chronic sinusitis are still resulting in lively discussion and extensive experimental investigation. There is no general agreement as to diagnosis, or indications for procedures which will best secure alleviation of symptoms. It seems probable, therefore, that there is no clear concept of the etiology of the disease, the anatomic or pathologic conditions present which militate against recovery, or the general surgical principles on which to base the attack on the condition; for, as Ballance[1] said, when there is a clear understanding of the pathologic conditions involved, there should be general unanimity as to the procedures necessary to secure the desired result.

In the last two decades, appreciation of certain physiologic factors has simplified the problem of differential diagnosis between chronic sinusitis and conditions that simulate it. The physiologic viewpoint in the problem of chronic sinus disease has been stressed by Lillie[2] for many years and has resulted in outstanding work on nasal physiology by his pupils Heetderks,[3] Stark,[4, 5] Hilding[6, 7, 8] and Hansel.[9, 10] Proetz[11, 12, 13] and others have given their attention to

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nasal physiology with the addition of much valuable knowledge as to many of its mechanisms. Mention of these names should be sufficient to recall their contributions and the effects they have had in revolutionizing the diagnosis of sinusitis, and medical and surgical treatment. This work has demonstrated that the foundation of a clear understanding of the problem of diagnosis must rest on appreciation of the physiology and structure of the nose and nasal mucous membrane. The function of the whole mechanism of the nasal chamber is to condition the inspired air, and alterations in the mucosa or the vasomotor control mechanism will readily produce symptoms which may be confused with sinus disease. The nose is normally most efficient in resisting and controlling infections. For instance, it has been found very difficult to infect a normal sinus by the injection of cultures of virulent bacteria into it. This resistance may be owing to the impenetrable mucous blanket and to the rapidity with which it is carried away by ciliary action, the presence of lysozyme in the glandular secretions, or the response of the reticulo-endothelial system to infection and to other factors. Therefore, to produce sinusitis, special conditions are necessary. Hypothetically the conditions necessary to produce sinusitis are: (1) interference with the vasomotor control mechanism that leads to anemia of the membranes and changes in the mucous blanket; (2) interference with ciliary movement by bacterial toxins, drugs or drying; (3) mechanical interference with the pathways of discharge due to abnormal structure and (4) unusual stress placed on the defense mechanism by unfavorable environmental conditions.

DIFFERENTIAL DIAGNOSIS OF SINUSITIS

Of the important conditions to be distinguished from chronic sinusitis, that of vasomotor rhinitis is sometimes difficult because of the associated migraine or erythrothermalgic type of headache with tenderness of the face and head which may accompany the nasal discharge. Unfortunately, the appearance of the nasal mucous membrane may not be of assistance. The classical full, boggy, mucous membrane, grayish in color, covered with watery exudate is simple to recognize, but does not correspond to the picture associated with many cases of vasomotor rhinitis. An allergic thickening of the sinus mucosa may also confuse the roentgenologist. A careful history, a microscopic study of the secretions, a diagnostic lavage, and observation of the patient for sufficient time should be enough to establish the diagnosis. When the condition is sufficiently severe to cause polypoid degeneration of the nasal membrane and when, due to interference with drainage and with ciliary activity, a secondary

infection has been set up, the problem of differential dignosis becomes truly difficult. This is the condition formerly designated "hyperplastic ethmoiditis." It is important to recognize that both allergy and infection are present, that allergy is the basic condition and that infection nearly always is secondary. Bacterial allergy is so infrequent that it can be virtually ignored.

Hypersecretory rhinitis, sometimes called physiologic hyperactivity of the nasal mucosa, a condition which usually occurs near the onset of puberty but which is produced easily by environmental conditions which throw great stress on the mechanism of the nose, is often confused with sinus disease, especially if contact headache is produced by swelling of the membranes. Although this error in diagnosis is due to mere carelessness, it is not infrequently made.

Atrophic rhinitis and ozena may be confused with chronic sinus disease because of the thick secretion and crusting that may involve the paranasal sinuses as well as the nasal chambers proper. A history of early onset of symptoms associated with menstrual disorders, faulty development of secondary sexual characters and the like, should be very useful in the case of ozena, even when the ozena is accompanied by the characteristic odor or when the full-blown crusting has not developed. Among older persons, the appearance of the nose taken in conjunction with the history of a late appearance of symptoms should be a sufficient guide to allow a distinction to be made between senile atrophy and chronic sinusitis. The beneficial effect of estrogenic hormone used locally should help to decide the question if doubt still exists. Abnormalities in other hormonal secretions, especially those of the thyroid gland, may produce symptoms of nasal congestion and increased discharge which may be confused with chronic infection in the paranasal sinuses.

The chronic swelling and discharge caused by the use of nasal inhalants, sprays, and nasal lavage may be a source of confusion in diagnosis unless the patient is directed to stop all medication during the course of examination. The effect of even long-continued abuse of the nose is usually abated in a week's time unless the abuse has taken the form of watery lavage, when the atrophic changes produced by a long course of this mistreatment will be found to be irreversible.

SYMPTOMS OF CHRONIC SINUSITIS

One might say, in general, that pain is the exception rather than the rule is cases of chronic sinusitis, and that unless there is an exacerbation of chronic sinusitis, the proffering of pain or headache

as a chief complaint should weigh against the probability of the presence of chronic sinus disease. The most prominent local sign of chronic sinusitis is a persistent nasal and postnasal discharge. Diurnal variations may be present which correspond to the optimal position for drainage of the sinus involved. Chronic sinusitis, however, is frequently without sufficient local symptomatology to bring itself to the notice of the patient. General symptoms of chronic sinusitis are most important to recognize from the standpoint of the well-being of the patient. A general rundown condition is one of the most frequent results of a chronic suppurative focus. This is a condition which is difficult to describe adequately, but is easily recognized by anyone with a moderate degree of clinical experience. Recurring respiratory infections may also depend on chronic sinusitis, recognized or unrecognized. Chronic bronchitis will be found to depend on chronic sinus infection in many cases. Arthritis is also occasionally found to originate from a sinus infection but, in my experience, sinusitis cannot be considered the primary cause.

The absence of pain or discomfort is notable, especially in the so-called silent type of sinusitis. In this type of sinusitis, the patient is usually referred by a medical colleague in an attempt to discover a cause in the nose and throat for a persistent and apparently baseless cough, a focus for fibrositis or arthritis, or occasionally for a rundown state of the general health. The patient may be entirely free from complaints referable to the nose or sinuses, and inspection of the nasal chambers or choana may not reveal any suspicious secretion. Only roentgenologic examination may reveal an opaque sinus which may prove to be the source of the disorder.

Chronic maxillary sinusitis, in about 25 per cent of cases due to extension from an apical abscess, is the most frequent singly-involved sinus in a chronic suppurative process. The most frequent complaints are an unusual tendency to the development of coryza, a unilateral discharge, a postnasal drip, or the sensation of unusually long teeth on the involved side. An occasional foul odor may also be a clue to a chronic maxillary sinusitis. Frequently inspection of the nasal chambers will reveal congestion of the mucosa on the involved side, and may reveal pus in the middle meatus. Pain in cases of chronic maxillary sinusitis may occasionally be complained of over the eye on the affected side. This is apparently due to the weight of secretions pressing on the antral floor.

The ethmofrontal group of sinuses are usually involved together and the tendency to overlook ethmoid involvement in disease of the frontal sinus led Jansen[14] to suggest in his original paper that

the condition of the ethmoid sinuses is the key to the condition of the frontal sinuses. The symptoms are frequently no more than a profuse purulent discharge on the side of the nose involved with, possibly, a vague sense of discomfort in the frontal region. If, however, a multilocular type of frontal sinus is present, pain may be severe due to complete closure of the opening into one of the divisions of the sinus and may recur at comparatively regular intervals, coincidental with the breaking through of the discharge and its gradual reaccumulation. Pus may rupture through the outer table with the formation of a subperiosteal swelling, and later a discharging sinus, or pus may rupture through the floor of the sinus anterior to the meridian of the globe and form a subperiosteal swelling in the anterosuperior aspect of the orbit forcing the globe downward and lateralward. Rupture may occur posterior to the globe and produce proptosis. Erosion of the internal table of the frontal sinus may occur with the formation of an epidural abscess or encephalitis of the frontal lobe. Meningitis is a comparatively rare complication of chronic fronto-ethmoidal sinusitis, due to the slow evolution of the lesion which usually seals off the arachnoid space.

Mucocele of the frontal sinus or, if secondarily infected, pyocele, may result from complete closure of the frontonasal opening resulting in a slow accumulation of mucoid secretion which gradually displaces and absorbs the bony walls of the sinuses. The only symptoms produced are the deformity and occasionally diplopia from displacement of the globe. They must be distinguished from neoplasms, but this is usually readily done by means of roentgenologic examination.

Chronic involvement of the spheno-ethmoid group of sinuses is the only condition with which pain is frequently associated. Owing to the close proximity of the sphenopalatine ganglion, irritation of this structure is not uncommon with a resulting sphenopalatine ganglion type of neuralgia. Generalized neuritic pains seem to be more common with involvement in this region than in any of the others. The discharge drops directly into the throat, and occasionally a postnasal drip may be the only symptom present in this group of cases. An occasional foul odor in the nose may also be the only sign of spheno-ethmoid involvement.

Involvement of the ethmoid labyrinth alone has been a very uncommon condition in my experience. For instance, in investigating 100 cases of infectious arthritis for sinus disease, we were unable to find a case of isolated ethmoiditis, although, according to published reports, one ought to expect a comparatively high incidence of "silent" ethmoiditis in cases of arthritis.

EXAMINATION OF THE PATIENT

In examining a patient who is suspected of having chronic sinusitis, the examination should proceed according to a well-considered routine, the most important step of which, I repeat, is a well-taken history. Inspection of the nose should include observation of the vestibules and alae before the insertion of the nasal speculum. Undeveloped alae, particularly, furnish the clue to many nasal complaints which the patient may ascribe to chronic sinus disease. Inspection of the nasal chambers with a nasal speculum is of the greatest importance. Irregularities of the septum should first be noted and then an attempt should be made to estimate the physiologic condition of the nasal mucosa. Valuable hints as to the nutrition of the individual and the function of the endocrine glands may be secured from the presence of a pale, boggy or shrunken membrane. The type of secretion seen and the presence of polyps may be suggestive of an allergic background. The nose should then be thoroughly cleaned and gentle suction should be applied. If pus appears, the regions in which it is first visible should be noted. The nasal mucous membrane should then be anesthetized and shrunken and the patency of the ostia of the maxillary, frontal and sphenoid sinuses should be tested with a probe. If open, the sinuses may be canalized and the type of secretion withdrawn noted. If the findings are suggestive of allergy, a smear of the nasal secretions may be examined for the type and number of leucocytes present. If chronic sinusitis seems a probable diagnosis, a radiograph should be taken. A diagnosis on the basis of roentgenologic findings alone is occasionally made, and may account for the lack of good results following some operations on the sinuses. The roentgenograph should be studied by the rhinologist himself for evidence of anatomic configuration of the sinuses, evidence of atrophy or hypertrophy of the bony walls of the sinuses, and evidence of destruction of the walls of the sinuses, in addition to evidence of thickened membrane or the presence of secretion in the sinuses. Exceptionally, particularly when the extensions of the sphenoid sinus are uncertain, lipiodol inserted into the sinus may be of diagnostic aid. Tomographs or laminographs may infrequently reveal evidence of a pathologic process not shown on ordinary simple or stereoscopic examination.

After roentgenologic studies, if pathologic change is indicated in the antrums which was not revealed by suction through a cannula, puncture and lavage of the maxillary sinus should be carried out. We feel that the results of lavage through the natural ostium cannot be depended on due to the frequent small size of the ostium of the

sinus. The nasal chamber should be inspected again after a period of thirty minutes to observe the appearance of pus in other portions of the nose. If pus is observed, its source should be identified. Shrinkage with Dowling packs and suction should be repeated on several occasions until the observer has satisfied himself that he has identified the source of the suppuration. Inspection of the spheno-ethmoidal recess both with cannula, pharyngoscope and postnasal mirror should be practiced routinely as infection in the posterior ethmo-sphenoid group of sinuses is most frequently overlooked. Such refined diagnostic methods as cytologic examination of the secretions suggested by Sewall[15] are of little help as they tell us nothing as to the site of the disease.

In cases of so-called silent sinusitis, such careful examination alone will reveal the sinuses affected, but in the obvious cases of chronic sinusitis, especially with rupture through the outer table or into the orbit, such meticulous examination may be unnecessary. In cases of pyocele or mucocele of the sinuses, displacement of structures and roentgenologic examination are alone of diagnostic value.

TREATMENT OF CHRONIC SINUSITIS

When confronted with a case of chronic sinusitis, the answer to the question, "Why has this sinusitis become chronic?" often will supply a solution to the problem of what treatment to adopt. Some abnormal structural development may interfere with free discharge, either in the septum or in the walls of the sinuses themselves. The deeper layers of the mucosa in the sinuses may have become the seat of disease to such an extent that the defense mechanisms of the body merely maintain the status quo rather than attempt to expel the invading organism. A condition that interferes with ciliary activity, such as allergic edema of the mucosa, may be the basis of the disorder; or disturbances of the endocrine glands may produce changes that interfere with the natural physiologic activity of the nasal chambers and sinuses. There are, of course, cases in which the cause of chronicity is too subtle for our analysis and yet, in most cases, consideration of the possible cause will point to a probable answer.

The problem of allergic swelling of the membranes is one which until lately defied us in our attempts at relief in many cases in spite of rigorous investigation of cutaneous sensitivity, elimination diets and the like. In the hands of even the most painstaking and competent allergists, disappointment was the rule rather than the exception. The recent work of Prickman[16] on the effect of histaminase in reducing the indolent type of allergic congestion, such as we see

in cases of so-called hyperplastic sinusitis has opened up a therapeutic vista of great promise for this condition. Two to four tablets of histaminase taken before each meal will often produce the remarkable effect of causing such a disappearance of polyps and reduction of edema that simple measures such as shrinkage with the Dowling[17] pack followed by gentle suction[18] will effect a cure of the secondary infection of the sinuses. Of course, the taking of histaminase must be continued over a period of unknown duration. Reduction of the allergic swelling of the membranes in this type of case is produced more than half the time.

Vaccines have been suggested for stimulating the natural defense mechanism to greater effort in those cases in which disease of the deep layers of the mucosa seem to be responsible for the chronicity. In my own experience, a few good results have been obtained by the use of an autogenous vaccine of the oral type as suggested by Besredka.[19] This vaccine in suitable dosage is dropped into the nasal vestibule, is allowed to run backward to the throat and is then swallowed. One difficulty with the use of autogenous oral vaccines is an occasional unusual activity so that an almost incredibly great dilution must be used to prevent unfavorable reaction. If any reaction such as fever or local pain is produced by the vaccine, it must be diluted until none occurs, lest the patient's condition be made worse instead of better.

Patients on an inadequate diet, which occurs more commonly than was formerly believed, will occasionally show great improvement of a chronically infected sinus if such dietary faults are corrected. It has been my experience that the alleged benefits of a favorable climate in effecting a cure of chronic sinusitis are grossly exaggerated. Many patients are seen whose sinus disease originated in such supposedly favorable places, and this tends to destroy belief in the curative effect of climate. Physiotherapy and roentgen therapy both seem to be unable to perform that which their proponents have occasionally claimed for them in respect to chronic sinus disease.

Repeated lavage of an antrum or of another sinus in at attempt to cure a chronic infection seems to be an entirely illogical procedure. In certain instances, such as in the case of an individual who, following an acute infection, has an antrum still filled with inspissated mucoid material, one or two cleansings will produce permanent relief from symptoms, but this type of case does not truly fall into the classification of chronic sinus disease with its characteristic pathologic changes. It is probably true that lavage through the natural ostium is sufficient to give relief in such a case. If bronchiectasis is

present, the coughing up of purulent secretion into the nose may be a factor in maintaining infection there, and measures directed toward lessening the infection in the thorax will help greatly in the care of the infected sinus.

SURGICAL TREATMENT

The elimination of mechanical factors that obstruct drainage, such as a deflected septum and spurs, should be done in many cases before any surgical procedures are attempted on the sinuses themselves. It has occasionally seemed that the negative pressure produced by inspiration must be a more important factor in securing emptying of the sinuses than is generally believed because of the frequency with which the sinuses that empty into the wider portion of the nasal chamber seem to be chronically infected in instances of septal deflection. This can hardly be due to the inhibitory effect of drying on ciliary activity because of the lack of an effect of this sort on the membrane in the interior of the sinus. The removal of severely diseased tonsils sometimes produces sufficient shrinking of the nasal mucosa to allow a chronically infected sinus to recover. In the case of an isolated chronic infection of the maxillary sinus, attention should be paid to the condition of the teeth, and if periapical infection is present, it should be eliminated.

If we have exhausted our magazine of medical and adjunct surgical treatment, or if the condition is one which obviously will not respond to such management, we are faced with the decision as to which type of surgical procedure to adpot for the condition at hand. Each case of chronic sinusitis must be individualized and considered on its own merits and the competent rhinologist must be provided with a sufficient variety of technics to take care of all possible variations in the expression of the disease. The words radical and conservative should have no place in the vocabulary of the rhinologist, for they have no real meaning as commonly used. The terms adequate and logical should be substituted, where necessary. Before embarking on surgical treatment of the sinuses, the rhinologist should try to discover certain fundamental surgical principles to guide him in the performance of his task. The first principal which we have evolved for our own guidance in operations on the paranasal sinuses is that all surgical procedures should be done under direct vision; blind instrumentation should never be attempted. The second is that adequate exposure, to reach the farthest extension of disease, should be secured so that all disease processes can be eliminated. The third is that drainage should be instituted in so far as possible along normal channels. The fourth is that multiple, rigid-walled cavities should be

eliminated and that the sinuses should be reduced to the largest single cavity or cavities that it is possible to obtain. The last is that the nose should be left, as nearly as possible, in a physiologic condition, and that to this end all functioning tissue possible should be preserved.

ANESTHESIA

Proper selection of the kind of anesthetic to be used and the method by which it is administered in operations on the sinuses are worthy of more attention than is often given to them. Local anesthesia has many advantages. Especially those of a relatively bloodless field, a decrease in the amount of blood lost during the operation, ease of administration, and freedom from annoyance due to the presence of the anesthetist near the field of operation. Were it not for certain dangers in its administration, there is no question but that local anesthesia would easily be the ideal method for sinus surgery. There is danger, however, of securing improper concentrations of the anesthetic and the danger of idiosyncrasy to the drug, but far outweighing these dangers is that of introducing infection into the deep tissues along with the anesthetic. This danger of infection arises from three sources. First, technical lapses in preparation of the solution to be injected. There are several opportunities in the preparation of the solution for slight breaks in aseptic technic which may lead to contamination of the solution. Second, in the preparation of the needles used for injection, particularly in the cleansing of their lumina and in sterilization, insufficient care may be taken. Particularly unfortunate in this regard is cold sterilization, so-called. This method can never be depended on to sterilize the lumen of a long needle. The third possibility for introduction of infection lies in the selection of routes of injection through the mouth or other regions which it is impossible to sterilize adequately, and careless technic in making injections into those regions which it is possible to sterilize adequately. Local anesthesia is particularly dangerous when subperiosteal infection, caused by invasion of the bony walls of the sinuses by the process of infection, exists.

Heilman[20] and one of us (Williams) have shown that there is some probability that the initial invader in cases of osteomyelitis of the frontal bone is an anaerobic streptococcus of slow growth and specialized cultural requirements, the staphylococcus, so often reported, being a secondary invader. The inclusion of adrenalin in the solution to be injected may furnish a more favorable condition for the development of anaerobic organisms, although experimental proof is not available. These dangers may be thought more theoreti-

cal than real, but it seems noteworthy that osteomyelitis as a postoperative complication of external operation, especially on the ethmofrontal group of sinuses, has assumed greater prominence with the increased use of local anesthesia. I have not found reports of osteomyelitis secondary to an external operation on the sinuses in any case in which local anesthesia was not used.

Of the general anesthetics, avertin can usually be depended on for basal anesthesia only, and must be supplemented by local or general anesthesia of some type. The newer intravenous types of general anesthetics are definitely contraindicated when there is the possibility of the embarrassment of the airway by blood or secretions such as exists in operations on the sinuses. The preferred type of a general anesthetic is a combination of nitrous oxide and ether given through an intratracheal tube. This requires a minimum of the anesthetic, an open airway is always maintained, forced respiration may be instituted if necessary, and the anesthetist is distant from the field of operation. The disadvantage of a general anesthetic lies in increased bleeding, which may obscure the region of operation, and an increased loss of blood, which may reach dangerous proportions in an extensive, especially a bilateral, operation.

ANESTHESIA FOR INTRANASAL PROCEDURES AND FOR EXTERNAL OPERATION ON THE MAXILLARY SINUSES

The use of the Sluder-Painter[21] technic combined with nerve blocking seems the most suitable for intranasal operations on the paranasal sinuses, for deep injections through contaminated regions are not required and the regions into which the injections are made are not in venous communication with the diploe of the frontal bone. Five grains of cocaine flakes moistened with just sufficient 1:1000 epinephrine to produce a solution are taken up on two applicators, one of which is placed anteriorly in the roof of the nasal chamber between the septum and the anterior tip of the superior turbinate. This secures anesthesia of the anterior ethmoid nerve branches as they enter the nose. The second applicator is introduced beneath the posterior tip of the middle turbinate and anesthetizes the branches from the sphenopalatine ganglion as they enter the nose. A cotton pledget wrung out of a 10 per cent solution of cocaine and placed beneath the inferior turbinate will interrupt sensation in the nerves coming through the anterior palatine foramen and supplying the antronasal wall inferiorly. This anesthesia is usually complete in ten minutes and will suffice for the majority of intranasal procedures in the average individual. For operation in the posterior part of the

nose this anesthesia should be supplemented by injection of the second division of the fifth nerve. This is easily accomplished by palpating the mandibular notch just below the zygoma and approximately 3 cm. anterior to the temporomandibular joint. A needle thrust directly through the notch will meet the lateral pterygoid lamina at a depth of 2 to 3 cm. The needle may then be withdrawn slightly and the tip directed 1 cm. forward and slightly upward to enter the pterygopalatine and sphenopalatine fossae. Two and a half cubic centimeters of a 2 per cent solution of procaine hydrochloride deposited at this point will produce anesthesia of the second division of the fifth nerve. If the tip of the needle passes through the sphenopalatine canal into the nasal chamber, the patient will notice the solution trickling down the throat. If an external operation on the maxillary sinus is to be performed, submucosal injection along the line of incision will be very helpful in lessening bleeding. In external operations on the fronto-ethmo-sphenoid group of sinuses, the danger of local anesthesia is more imminent as the field injected has venous communications with the veins of Breschet in the diploe of the frontal bone. For this reason general anesthesia through the intratracheal tube is preferred for such procedures.

POSITION OF THE PATIENT

For all intranasal procedures and for external operation on the maxillary sinus, the upright position with the patient sitting in a chair is preferable because of the lessened bleeding in this position. For patients who are particularly prone to faint, however, the semi-reclining position is preferable as no co-operation on the part of the patient is required. For external operations on the ethmofrontal group of sinuses the same reclining position is preferable because of the amount of surgical shock connected with such an extensive procedure on the sinuses, even when done under local anesthesia.

TREATMENT OF SHOCK

In major procedures on the sinuses, as in major surgery in other portions of the body, the prevention of surgical shock is of major importance. In these well-vascularized areas, particularly if the operation is done under general anesthesia, the amount of blood lost may reach 500 to 800 cc. Since loss of blood is the principal precipitating factor in surgical shock, methods of restoration or of preventing its loss offer the only really effective treatment. To be successful these methods must be started at the first sign of surgical shock, for if a period of prostration is allowed to intervene before

treatment is started, recovery becomes doubtful. It is of even greater advantage to prevent the appearance of even the first signs of shock, such as dropping blood pressure and clammy perspiration. Therefore, in external operations on the ethmofrontal group of sinuses, blood transfusion given during the course of the operation is an important adjunct to treatment, as it will usually prevent the development of this complication. Even though shock need not be anticipated, the better postoperative condition and the speedier recovery of patients who have received a transfusion during the course of the operation are noteworthy.

In major procedures, during which loss of heat and body fluids as the result of a prolonged period of anesthetization is anticipated, it is advisable to start the intravenous administration of fluids soon after the patient has been anesthetized. A physiologic solution of sodium chloride or a solution of 5 per cent dextrose in physiologic sodium chloride, given at the rate of about 5 to 8 cc. per minute, seems to be of definite benefit to the patient. The lost fluid is largely replaced and the blood pressure is supported. If an excessive amount of blood is being lost, the administration of the solution of sodium chloride or the solution of glucose and sodium chloride should be discontinued and citrated blood should be administered through the same intravenous set. The rate of administration and amount of blood required depend entirely on the amount of blood lost and on the condition of the patient.

A 6 per cent solution of acacia and normal saline solution with or without vasopressor drugs may be used to avoid shock if blood is not available.

OPERATIONS ON THE MAXILLARY SINUS

Operations on the maxillary sinus are performed through two routes; that through the canine fossa, originally suggested by Caldwell,[22] and the operation through the inferior meatus of the nose originally suggested by Mikulicz.[23] In the modification of the Mikulicz operation proposed by Hempstead,[24] the anterior attachment of the inferior turbinate is incised, the turbinate is infracted upward to expose to direct vision the naso-antral wall. This is then broken through with a Wilhelminski antrum perforator and the opening is enlarged posteriorly to the ascending process of the palatine bone and forward to the hard bone of the pyriform opening, inferiorly to the floor of the sinus and superiorly to the attachment of the inferior turbinate, care being taken anteriorly not to injure the nasolacrimal duct. Through the opening thus secured, the entire interior of the

maxillary sinus may be inspected except for small areas anteriorly and superomedially which may be inspected with the aid of a naso-pharyngeal mirror. Granulation tissue, polypoid degenerations, and even cysts, can be removed cleanly and without difficulty. Packing is not placed in the antrum unless bleeding makes it necessary. The Caldwell approach is indicated (1) when it is imperative to secure a good view of the entire interior of the sinus; (2) when a malignant lesion is suspected of being present; (3) when the naso-antral wall is too thick to allow an adequate window to be made endonasally or (4) when bony walls or partitions exist in the sinus must be removed to meet the requirements of the surgical principles which I have adopted.

In the Caldwell[22] technic, an incision in the buccolabial fold is made from the frenum to a point about 1 cm. lateral to the root of the canine tooth. The periosteum is then elevated along a line extending from the pyriform opening to a point lateral to the root of the canine tooth. Care must be taken in elevating the periosteum upward not to disturb the infra-orbital nerve in its exit from the foramen, as this is likely to result in an annoying neuralgia. The maxillary sinus is then entered through the canine fossa and the opening is enlarged sufficiently to allow adequate inspection of the interior of the sinus. Grossly diseased portions of the membrane may be removed and bony septa, if present, may be ablated. The bony naso-antral wall beneath the middle turbinate is next removed. The mucous membrane may then be incised and cut away. The buccal wound may be sutured if desired. The advantages of a vertical incision over a horizontal one in making the initial incision are probably exaggerated, as numbness of the central incisors is the rule even after the intranasal operation and it depends on interrupting the dental nerves in the naso-antral wall rather than in the anterior wall of the sinus. If thought desirable, the entire thickness of the bone between the pyriform opening and the canine fossa as suggested by Denker,[25] may be removed. Tales of weakening of the upper jaw or of annoying discharge from the nose following employment of the Denker technic seem wholly apocryphal.

In both the intranasal and external technics, as much of the lining membrane of the sinus as possible should be preserved, even when it appears thickened and the seat of disease. If adequate drainage is secured, these apparently diseased portions of the membrane will return to normal. In either technic, the final result of the operation depends on the adequacy and patency of the endonasal opening that is secured.

If a fistula through the alveolus to the antrum exists, it must be closed before a cure of the disease in the maxillary sinus can be expected.

Both of these technics are physiologic in that no functioning tissue in the nose is destroyed if care is taken not to disturb the anterior end of the inferior turbinate, which plays an important part in directing the inspired air upward into the middle meatus.

OPERATIONS ON THE FRONTO-ETHMO-SPHENOID GROUP OF CELLS

It is in isolated involvement of the ethmofrontal group of the paranasal sinuses that the question is most frequently raised whether the intranasal or the external approach to the involved cell groups is the most adequate and logical. If the disease has broken through the frontal or the orbital wall of the sinus, the decision in favor of some type of external operation is easy to reach. In case there is no such easily recognized signpost, the decision must be reached by considering and giving proper weight to the several factors that will influence the final result.

The first of the factors to be considered is the anatomic structure of the frontal sinus. If the frontal sinus is very extensive and of great depth anteroposteriorly, the probabilities that an adequate intranasal opening will be sufficient to secure a cure is great. If the anteroposterior distance is small, there is greater possibility that in the shallower sinus, drainage from the lateral extensions of a large sinus will be interfered with. If the sinus contains septa that divide it into two or more loculated collections of pus, the probability is that an intranasal approach will not result in sufficient drainage to afford a cure. The less the frontal sinus extends laterally and upward, the greater the probability that intranasal drainage will effect a cure. If the intersinus septum can be seen to contain an infected cell, the probability again becomes great that whatever the size of the sinus, intranasal drainage will not cure. The space between the wall of the orbit and the intrasinus septum, in other words the width of the ethmoid labyrinth, is worthy of attention. If this space is wide, allowing easy instrumentation, it will be much easier to obtain an adequate frontonasal opening than if this space is narrow. The extent to which the ethmoid cells extend laterally in the roof of the orbit is of great importance in forming a decision, because it is obviously impossible to reach these lateral extensions of the ethmoids when they exist, by any type of intranasal approach. It is important to note the amount of sclerosis of the bony walls of the sinuses, be-

cause too great an increase in the thickness of the dividing septa requires so much force to break them down that operations by the intranasal route become increasingly dangerous. Previous intranasal surgery is likely to have caused a marked increase in the amount of bone sclerosis, and it is probable that if an intranasal approach has once failed to achieve its object, this type of approach should not be repeated; an external approach should be chosen in its stead. The serious complication of the external operation is the development of a spreading osteomyelitis of the frontal bone. This complication, however, usually follows operations done under local anesthesia. The condition seems to start in the cancellous bone of the nasion and advance backward to the cancellous bone of the frontal bone by thrombophlebitis of a large, comparatively constant vein of Breschet which comes down to the region of the nasion from each side. One of the advantages of the Killian[26] type of procedure is the interruption of this venous communication.

If the middle turbinate has been so destroyed by disease or previous operation that retention of its remnants would not be physiologically useful, it makes reaching the lateral extensions of the ethmoids intranasally somewhat easier, so that this might tend to exert an influence toward an intranasal approach. If the sinuses are being operated on to clear up a focus of infection, as in a case of arthritis or bronchiectasis, it is injudicious to attempt an intranasal approach when one cannot afford to leave even the smallest infected cell behind to continue infection.

The type of intranasal fronto-ethmoid exenteration which is the most physiologic, is one which leaves the middle turbinate intact enough to function at the completion of the operation. This technic was first suggested by Pratt[27] and it must now be of considerable satisfaction to him to find his method substantiated by the recent investigations of Hilding, Proetz and others. Entrance is made into the ethmoid labyrinth through the bulla ethmoidalis and sufficient bone is removed to enter the frontal sinus directly above. The opening is then enlarged forward and an attempt is made to remove the internal nasal spine. The opening is then carried medially as nearly as possible to the intrasinus septum and septum nasi. The posterior wall of the frontal sinus is then palpated and the shelf of ethmoid cells projecting forward below the frontal sinus is removed. The opening secured should be sufficiently large to admit a 1 cm. Faulkner ring curette. The lateral and upward extensions of the frontal sinus may then be explored with a curette and any grossly diseased tissue removed. The removal of the ethmoid labyrinth should then

be proceeded with, remembering that the roof of the ethmoid labyrinth rises into the frontal bone 1 to 2 cm. above the level of the cribriform plate. The operation should not be terminated until it can be observed that the roof of the labyrinth has been reached and is clean. During the course of the operation any concealing blood must be wiped away and no motion should be made which cannot be adequately observed. There can be little question that an adequate intranasal operation requires more skillful and delicate technic and is a more dangerous procedure than an external approach. When well done, however, it can be a completely satisfactory procedure in a favorably developed ethmoid labyrinth.

The external operation on the fronto-ethmoid group of cells can be divided into three types. The first, in the order of their evolution, that of Jansen[14] in which the floor of the frontal sinus is removed and tissue that is the seat of disease removed from below. The second is that type suggested by Killian[26] in which, after removal of the floor of the sinus, a bridge of bone is left to preserve the contour of the superciliary arch and the anterior wall of the frontal sinus removed above it, obliterating the upper portion of the frontal sinus. The third type suggested by Riedel[28] is that in which the inferior and anterior walls of the frontal sinus are completely removed with the lining membrane in its entirety so as to obliterate the sinus. This progression from a less to a more mutilating procedure is obviously owing to the difficulties experienced with recurrences of infection in the sinus previously operated on. There can be little doubt that this technical problem has been very difficult for the rhinologist to solve. Anderson[29] found that failures to cure suppuration in the fronto-ethmoid group of cells depend first on incompleteness in operating, especially failure to eradicate disease in the lateral extensions of the frontal sinus or in cells in the intrasinus septum or in finger-like extensions of the sinus into the frontal bone superiorly, and second, closure of the frontonasal opening. To explore all the extensions of a large sinus, adequate exposure is required and therefore the small incision medial to the inner canthus suggested by Sewall[30, 31] and later advocated by Smith[32] is contraindicated, except in very small frontal sinuses in which the propriety of the external operation might be questioned. The original incision of Jansen being virtually an extension of the Sewall incision into the eyebrow carried laterally as far as necessary aids greatly in exploring those lateral extensions which extend occasionally into the zygoma itself and which it is so necessary to eradicate to obtain a cure of the suppurative condition in the sinus without adding any obvious scar.

The incision should be started low enough so that the lacrimal fossa can be easily exposed and carried into the brow from below upward and outward to avoid cutting the hairs of the eyebrow. Cross hatchings in the skin along the line of incision will be useful in closing the incision. The periosteum is then freed from the lamina papyracea and the floor of the frontal sinus. Bleeding vessels, including the anterior and posterior ethmoid vessels, are best controlled by coagulation with diathermy, as this avoids burying quantities of foreign material such as sutures in the wound. The frontal sinus is then opened with a gouge just lateral to the nasal process of the frontal bone. This incision should be made with some care because of the anomalous forward bulging of the posterior wall of the frontal sinus occasionally encountered in this region, as it is inadvisable to perforate the dura of the frontal lobe. After the opening has been made in the bone it should be enlarged downward to the lacrimal fossa, laterally to the farthest extent of the sinus, including removal of the lateral angular process of the frontal bone if necessary, and carried backward to remove the entire bony floor of the frontal sinus and the floor of any orbital-ethmoid cells which may be present. The frontal sinus and orbital-ethmoid cells should be completely cleaned of diseased tissue and bony septa interfering with drainage from the cavity should be removed.

If the anteroposterior dimension of the frontal sinus is too slight to allow adequate removal of disease, or if the bony septa are too sturdy to be easily removed, the maneuver of Killian may be resorted to. The presence of disease such as localized osteomyelitis of or fistulous tracts through the anterior bony wall of the sinus also makes this technic desirable. To accomplish it, the integument is raised from the forehead over the frontal bone, care being taken to leave the periosteum attached to the bone for the sake of its nutrition. If necessary, to secure exposure when the disease is unilateral, the cutaneous incision may be extended over the root of the nose in the way in which the two frontal incisions are connected in cases of bilateral disease. Deformity is lessened and the nutrition of the bridge of bone furthered if it is made as broad as possible. The periosteum is cut through mediolaterally 2 to 2.5 cm. above the most superior portion of the frontal incision. The periosteum above the line of incision is then elevated to a point above the highest extension of the frontal sinus. Starting just above the edge of the attached strip of remaining periosteum, the anterior wall of the frontal sinus is entered with a gouge and is removed, leaving that portion covered with periosteum as a bridge to maintain the contour of the forehead. A very important step, first suggested by Lillie,[333]

consists in bevelling the bone edge from the upper limit of the sinus into the frontal bone so that in bringing down the cutaneous periosteal frontal flap a pocket is not formed at the edge of the sinus. This bevelling is most easily accomplished with an Alexander gouge.

The fashioning of the frontonasal opening must next be attended to. This is the most important step in the Jansen and Killian technics, as Anderson pointed out that the principal cause of failure to cure suppuration in the frontal sinus was closure of the frontonasal opening. Lillie[33] pointed out the importance of preserving the frontal process of the superior maxilla, as this bony shelf prevents the closing of the frontonasal duct by the collapsing inward of the soft tissue in this region. The internal nasal spine should next be removed. This is most easily accomplished by removing it with a gouge placed at the edge of the frontal process of the superior maxilla directed downward and medially. The removal of the medial part of the floor of the frontal sinus is carried over to the septum and back to the posterior wall of the frontal sinus and the beginning of the ethmoid labyrinth by the use of Lillie's modification of Kerrison's mastoid rongeur, and with this instrument the agger nasi cells are also exenterated. This is done by infracting the middle turbinate laterally, opening the ethmoid labyrinth through the bulla by the intranasal route and removing sufficient of the ethmoid labyrinth to enter the frontal incision. The Kerrison-Lillie rongeur is then introduced through the nose and the removal of bone is accomplished with careful preservation of the middle turbinate. If both frontal sinuses are involved, better maintenance of the frontonasal opening is secured if the intrasinus septum is removed and its removal is carried down into the septum nasi as suggested by Lothrop. Lillie[2] has pointed out that the Lothrop[34] maneuver is also valuable in cases in which an operation has been previously performed if the previous surgeon has destroyed the frontonasal passage by removal of the frontal process of the superior maxilla. The opening of a healthy frontal sinus has not been observed to produce complicating infection.

Exenteration of the ethmoid labyrinth may next be performed if this region is involved, keeping in mind that the roof of the labyrinth rises often 2 cm. above the level of the cribriform plate and is often very thin and may be deficient in bone, especially anteriorly. On reaching the anterior wall of the sphenoid, its natural ostium may be identified with a probe; this is enlarged. The anterior wall of the sphenoid may then be removed down to the floor with the Kerrison-Lillie rongeur. Care should be taken to remove the bony

partition between the posterior ethmoids and the lateral portion of the sphenoid sinus. In doing this, any extension of the posterior ethmoid cells into the body or alae of the sphenoid will be discovered if searched for. In this procedure of removal, particular care should be taken not to remove completely the posterior attachment of the middle turbinate; a hinge of mucosa is sufficient, however, to maintain it in position. It is usually unnecessary to remove the floor of the sphenoid sinus, but if this is desirable because of especially deep extensions into the pterygoid process, it may be done. The sphenopalatine artery may be grasped with a hemostat and may be coagulated with diathermy, the mucosa elevated from the undersurface of the floor and the floor is bitten out. Occasionally, the sphenoid floor is so thick as to require a lateral and medial cut with a chisel after which the bone may more easily be removed with a strong biting forceps. No advantage can be expected from removing normal mucosa from the sinuses, especially when it is so completely normal that previous to removal it must be loosened by gauze balls dipped in picric acid solution as suggested by Smith. Normal mucosa should be removed only when it lies in a region which may be closed off from the remainder of the sinus in healing, with a resulting mucocele. Before closing, the lamina papyracea should be removed to the roof of the antrum and any antro-ethmoid cells which may be present may be removed. The importance of this was stressed by Sewall.[30] The antrum, if diseased, may be opened from above and diseased membrane or bony partitions may be removed.

The Riedel procedure, that is, complete ablation of the sinus, is similar to that of Killian, save that the bridge of bone is completely removed. In this procedure, it is very important that no vestige of mucosa be allowed to remain; a mucocele will result if a vestige of membrane remains. If complete ablation of the frontal sinus is thought necessary, the resulting deformity may later be repaired by the insertion of cartilage implants to restore the contour of the forehead after a delay of six months to a year. In this procedure, of course, attempts to maintain a patent frontonasal opening are unnecessary, which is a real advantage, for, even with the best technic, exuberant granulations will form in this region which on organizing may close the frontonasal opening with dense scar tissue. Killian has suggested cutting and dissecting downward a flap of mucosa from the superior turbinate to the floor before commencing the external operation, which flap is later used to line the frontonasal opening. This is an entirely feasible but technically difficult procedure. Smith has suggested lining the frontonasal opening with

a skin flap, a procedure which would result in the production of a cholesteatoma in the sinus if the graft could be made to take. Fricke and one of us (Williams)[35] have previously reported a procedure which has given us great satisfaction, namely, the insertion of 9 gram minutes of radium in a brass and silver capsule into the frontonasal opening. This prevents the formation of granulation tissue and tends to prevent subsequent closure of the opening by scar tissue.

In closing the external incision, care should be taken to approximate the cut edges of the periosteum, as this is an additional help in the prevention of an inward collapse of soft tissues to close the frontonasal opening. Particular care should be taken to approximate the tissues in their previous position in the region of the trochlea of the superior oblique muscle because a tendency toward diplopia on looking downward is one of the disadvantages of the external operation, especially when done bilaterally.

INTRANASAL OPERATION ON THE SPHENOPOSTERIOR ETHMOIDAL GROUP OF PARANASAL PNEUMATIC CELLS

Intranasal operation on this group is best done by opening into the sphenoid first and exenterating the posterior ethmoid cells by working from the sphenoid; for, in this way, one is working forward and downward away from the dangerous region instead of upward and backward toward it. The posterior tip of the middle turbinate may be removed for exposure if necessary, but this structure should be entirely preserved if possible to prevent the inspired air reaching and dehydrating the lining membrane of the sphenoid which is supplied with few secreting mucous and serous glands. The natural ostium should be located and enlarged enough to allow the insertion of Lillie's modification of the Kerrison rongeur. The great mechanical advantage of this instrument allows bone to be removed with a minimum of muscular effort and therefore is always under perfect control. The anterior wall of the sphenoid is removed over to the septum, up to the roof and down to the floor of the sinus. The dividing wall between the sphenoid and posterior ethmoids is next engaged and completely removed to the lateral wall of the sphenoid. The posterior ethmoids are then exenterated with the same instrument, care being taken not to detach completely the posterior attachment of the middle turbinate. The region of the superior face of the body of the sphenoid is then explored with a probe to be sure that posterior ethmoid cells do not invade the body of the sphenoid. Care should be taken, in removing bone toward the

floor of the sphenoid, not to injure the sphenopalatine artery, as bleeding from this vessel is likely to be profuse. If this occurs, an attempt should be made to grasp the bleeding vessel in a tonsil forceps and coagulate it with diathermy. If this fails, a postnasal pack may be necessary.

POSTOPERATIVE CARE

If the operation on the sinus has been properly performed, there is more danger of meddlesome overtreatment than there is danger of failure to care for the case adequately. If it has been possible to avoid packing the sinuses, a minimal reaction should be present. The avoidance of packing also tends to diminish the possibility of intracranial complications. The field of operation should be completely avoided the first four days. When radium is to be inserted into the frontonasal opening the tube should be withdrawn and the radium inserted. Silk or dermal skin sutures should be removed on the third day to avoid scarring, the deep tension sutures being cut but left in place a day or two longer. If an intranasal operation has been done, secretions should be withdrawn by gentle cannula suction. Lavage of the sinuses should be strictly avoided, as Lillie has pointed out that the use of watery solutions in the sinuses not only prolongs discharge, but also produces discharge.

Sulfanilamide used postoperatively in extensive external operations on the sinuses seems to promote healing of the wounds and cessation of the discharge. This type of therapy should not be continued beyond the fourth day. On dismissal, patients on whom external operations on the fronto-ethmoid group of sinuses have been done should be warned to avoid exposing the forehead to cold winds as this tends to produce sudden edema of the lids and may produce temporary closure of the frontonasal opening. This tendency is present from six months to a year after operation.

CONCLUSIONS

A careful differential diagnosis is the most important step in the treatment of chronic disease of the paranasal sinuses. An operative technic which logically fits the individual case and which will be adequate to take care of the disease present should be selected.

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XXXIII

RETROPHARYNGEAL ABSCESS WITH REFERENCE TO ABNORMALLY LARGE PERCENTAGE OF ADULT CASES*

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When looked into, the seriousness and the high mortality rate evidenced in retropharyngeal abscess is appalling. So often, in examining hospital records and case reports the diagnosis "retropharyngeal abscess—incision and drainage, recovered or well" is met. Many of these cases are not true retropharyngeal abscesses, but rather supratonillar or peritonsillar abscesses. That is, their etiology and anatomy have to do directly with tonsillar and peritonsillar tissue, as differentiated from retropharyngeal tissue. It is with this latter group that we are concerned, and of which twenty cases are here reported as seen in this section of the country with their analysis.

Retropharyngeal abscesses have been recognized for ages, being reported by Galen in the second century. From the eighteenth century, numerous case reports fully descriptive and authenticated are on record, and yet, the seriousness is not fully brought to our attention until careful examination of case reports and articles published are gone into. In this paper are reported twenty cases with a mortality rate of thirty per cent. The admission diagnosis varied greatly; from "cross and fretful child," "convulsions" to "obstructive laryngeal dyspnea" and "diphtheria." It is surprising how little space and comment are devoted to this condition in many text books.

McKenzie's[1] mention of Bokai's cases (144) in 1876, is noteworthy, and especially the number (129) of cases reported as of idiopathic origin. William Cullen[2] (1801) writes "Of the Cynanche Pharyngoea," speaking as though the condition were one of continuity for "the inflammation of the mucous membrane often spreads upon the pharynx and into the beginning of the esophagus, and thereby renders deglutition more difficult and uneasy; but such a case does not require to be distinguished as a different species, and only requires that blood letting and other remedies should be em-

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ployed with greater diligence than in ordinary cases." He does not refer to the surgical treatment; never having seen a case himself, he refers to "practical writers who have taken note of such cases," and "the method of cure." "Cynanche" means severe sore throat with threatened suffocation, but as Cullen mentions difficult swallowing and no word of impairment nor embarrassment of respiration, I feel he had in mind retropharyngeal abscess.

Often cases are seen late when complications are present and consultation on, or examination of the patient is requested for other than retropharyngeal abscess. Such requests are noted as "Otitis Media", "Glands of the Neck", "Impairment of Respiration", "Fever and Anorexia of undetermined origin—Focus or Infection". When the dangers and complications attendant upon this condition are fully realized, then only will a more wholesome respect be given to it.[3] Several pediatricians have written excellent articles with case reports on the condition.[4] Frank[5] rightly says "any effort we can make to popularize the seriousness of the condition and its diagnosis, will be instrumental in relieving acute agony and, possibly, of saving life."

In my series, the ages varied from eight months of age to sixty years. There was practically no difference in the sexes, there being eleven males and nine females; there were eleven whites and nine negroes. There were thirteen children, ages eight months to ten years, and seven adults whose ages were 24, 25, 27, 33, 35, 39, and 60 years, respectively. There were three deaths among the thirteen children and three among the seven adults. I cannot say that in any case in this series did under-nourishment, or the presence or absence of tonsils, seem to play any part. The general health and appearance, especially of the children, were good; several of the patients came from the better walks of life. The condition was much more prevalent in children, seeming to have a distinct relationship to taking cold, the existence of the lymphatic nodes (Waldeyer's ring), and retropharyngeal glands. These latter mostly atrophy, or disappear, about the eighth to tenth year.[6]

Retropharyngeal abscess in the adult does not differ in its pathology, complications, nor treatment for that of the child.[7] It often does differ in its etiology, especially as regards tuberculosis and foreign bodies.

The anatomy and pathology of the retropharyngeal spaces and lymphatics have been fully described and minutely studied by Gillette,[8] Most,[3a] Wood,[9] Mosher,[10] Faier[11] and others. Lederer[6] remarks "The great vessels in this cavity, as well as its proximity to

the base of the skull and neck furnish a number of dangerous possibilities".

ETIOLOGY

Retropharyngeal abscess is much more prevalent in children.[12] Turner[13] states that fifty per cent of the cases are under one year of age; that it is rare in adults (not so in this series), and occurring more often in boys than in girls. DaCosta[14] held it to be an infectious process of the mucous membrane of the mouth and nasopharynx. The offspring of tuberculous or syphilitic parents,[32] those children with rickets, or those who have an inherited diathesis, are more prone to the condition.[15] Bokai,[1] Vas and many others,[5] have shown in their series how much more prone young children are to this infection, and the close relationship of the upper respiratory infections. Especially do head colds, rhinitis, sinusitis, nasopharyngitis, otitis media, Waldeyer's ring, wth the lymphatic drainage into the deep cervical and retropharyngeal lymph nodes, predispose the individual. Still another cause is the marked nasal blockage brought on by hypertrophied adenoidal tissue. Many cases of otitic origin have been reported.[16] In children upper respiratory infection is generally found[4] (Clark and Barysh) as is some mild pre-existing illness, colds, cervical adenitis, tonsillitis and sore throat.[17] Strictly speaking there seems to be no seasonal prevalence.

The condition is reported as much less frequent in adults. Frank[5] (1921) reports four cases, Christierson[7] (1933) five cases, Watson[18] (1903) one case and was able to locate only sixteen previous reported adult cases in the literature.

As causative factors in the adult, tuberculosis must be first considered.[19] "Any prevertebral shadow, even without visible bone erosion, represents bone detritus plus inflammatory reaction and is pathologically an abscess."[20] Tuberculosis, whether of vertebral caries or secondary glandular invasion, trauma by instrumentation,[21] fracture of the vertebrae, as in the case reported by Laszlo,[22] syphilis (gumma) as in Christierson's[7] case. Upper respiratory infections must be considered; especially in attacks of influenza; with the severe rhinitis, nasopharyngitis and sinusitis which may accompany it. Also, foreign bodies such as a spicule or splinter of bone imbedded in the hypopharyngeal wall. I have personally had one such case. A case in which an elongated styloid process was unknowingly fractured and broken loose while performing a tonsillectomy; later a retropharyngeal abscess on the left side developed, and upon being opened and evacuated the piece of styloid process was recovered. Also, one must bear in mind dental infections, decayed and abscessed

teeth and bone necrosis. I feel that many of the extensive luetic destructive lesions we see in the pharynx of the negro race must be preceded by abscess formation in an existing gumma.

SYMPTOMATOLOGY

The condition should be thought of somewhat differently in the child than in the adult as regards symptoms.[7] In the adult, the symptoms and signs all point to the cervical region, while in the child, who doesn't complain so quickly, the symptoms may lead us far astray unless we are on a close lookout.

In children most cases complicate acute infections of the upper respiratory tract and the onset may be quite insidious,[4] and definite signs few or even wanting, so that the disease is entirely overlooked.[23] There may or may not be an elevation of temperature, children frequently do not complain, or there may be a croupy cough, some interference with breathing and swallowing especially in a nursing child; the evidence of nasal obstruction if the abscess is located high, regurgitation through the nose, the dysphagia, odynphagia, dyspnea, "quacky" cry or altered voice "cri de canard" of Reigenier, stertorous breathing, and retraction of the head when the abscess is situated low are danger signs. The neck may be stiff or held rigid and the head tilted toward the uninvolved side. Inanition, anorexia, rapid loss of weight due to the lack of and inability to take food may be present. Pain, if any, is deep seated. There may be, and usually is when we see the case, swelling of the glands of the neck of one side or the other. The child may have convulsions, facial nerve paralysis or suppurative otitis media. The head is often thrown back at inspiration and a marked wheeze present if there is any impending dyspnea; the throwing back of the head aids in clearing the hypopharynx and in part allows passage of air to the larynx[24] If there is a marked hypopharyngeal swelling, this often leads to edema of the larynx and the cardinal signs of air hunger; obstructive laryngeal dyspnea.[25]The bursting or rupture of a retropharyngeal abscess may produce immediate suffocation. Also, there is considerable difficulty in expelling secretions[21] by coughing, swallowing or expectoration. When bloody mouth secretions are present in a child, we should immediately think of retropharyngeal abscess; and also that the condition may complicate a discharging ear[16] The symptoms and signs may all be explained on an anatomical and mechanical basis. Definite signs of abscess may be noted on inspection and palpation.[24] but remember the impending danger with the use of any force in opening the mouth, or with digital pressure; i. e., rupture of the abscess and death by suffocation. (Wendt)

In the adult the symptoms, signs, and the complaints all point directly to a pharyngeal condition.[7] Swelling of the neck, if present, altered voice with aphonia at times, marked discomfort on swallowing, some dyspnea, anxiety, inanition, nasal obstruction, deep seated pain, often extending into the shoulder and down the arm, especially if the abscess is a "cold one", i. e., of tuberculous origin with caries of the cervical vertebrae. Generally a leucocytosis and prostration are noted unless the abscess is due to tuberculosis or syphilis.[7] In the adult the onset is similar in most cases to that of quinsy from which it must be differentiated. The usually smooth, tense, rounded elevation with fluctuation back of the pharyngeal wall tells of the abscess.[26] The voice is muffled and lacking in nasal resonance, cough and expectoration is difficult. Generally the condition takes a week to ten days to develop, though as short an interval as four days from the onset is noted.[24] Edema of the glottic structures may be rapid, or if edema is present an acute exacerbation takes place with rapid signs of suffocation. The most common symptoms in the adult are pain, dysphagia, odynphagia, dyspnea, noisy breathing, snoring, nasal obstruction, anorexia, regurgitation and swelling of the glands of the neck.

DIAGNOSIS

Diagnosis is dependent largely upon the history, signs, symptoms (the anamnesis), inspection, mirror examination if low, palpation, x-ray and exploratory needle. The last is seldom if ever needed. In the case of a one year old boy, Jones, Brown and Fine noted the importance of x-ray.[27] Its importance was also noted in the cases of Christierson[7] and Fine.[27] Hay[28] in his book describes the x-ray findings in retropharyngeal abscess; and Sever[20] discusses the significance of x-ray findings, especially as regards tuberculous abscesses. Usually no difficulty is experienced in making the diagnosis once the symptoms and signs are manifested, but it is the complicated cases and the overlooked cases that lead to diagnostic difficulties and high mortality rate.

In differential diagnosis, several conditions must be borne in mind and some of the conditions may be the result of retropharyngeal abscess.

Edema of the larynx also may be a complication, as might acute pharyngitis, marked adenoidal hypertrophy and osteomyelitis. Then the conditions of aneurism, peritonsillar abscess, tonsillitis, croup, laryngeal diphtheria, foreign bodies, cervical scoliosis, malformations

and neoplasms must be figured on, and the differential points evaluated.

To be carefully considered is the condition of otitis media suppurative with marked cervical adenitis (glandular fever) complicated by retropharyngeal abscess.[11] Richards called attention to the symptoms produced when the abscess was high, giving nasal blocking, and when low, giving laryngeal obstruction.[24]

COMPLICATIONS AND PROGNOSIS

In the well defined, usually easily diagnosed case with prompt incision and drainage, the prognosis is good. However, when one examines the complicated cases the high mortality is astounding. Especially in the tuberculous type the death rate is high. Hemorrhage complicating retropharyngeal abscess should be looked upon as most grave, calling for immediate ligation of the external carotid or common carotid arteries or the jugular vein, as so well discussed by Lifschutz[29] who reports a case, and who found 23 cases reported in the literature; Richards[24] who reported three cases in his series, and Frank and Abt who report four cases of "severe arterial hemorrhage from the pharynx and middle ear", which were successfully operated upon and recovered.[30] In this present series, there were two cases of severe hemorrhage, both fatal. One was operated upon and ligation of the right common carotid artery successful, but was followed later by a fatal hemorrhage from the vertebral artery. The second case had a fatal expulsive hemorrhage, the abscess had been opened previously, while a spinal puncture was being done because of meningeal symptoms.

Other complications are spontaneous rupture with suffocation, two cases in this series. Laryngeal spasm, tracheal or bronchial erosion, convulsions, septicemia, metastatic abscesses, pneumonia, lung abscess, mediastinitis, and abscess rupturing into the middle ear (one case recorded in this series), or retropharyngeal abscess complicating otitis media as reported by Jones,[16] Faier,[11] Frank[5] and others. Mosher reports thrombosis of the jugular vein in a case of retropharyngeal abscess.[31]

TREATMENT

Whether the retropharyngeal abscess be primary or secondary, the treatment is surgical, except in cases of gumma. The opening should be intra-oral as a rule, save in cases of tuberculous abscess which should always be opened externally. They should be treated as tuberculous abscesses are treated in other parts of the body, but

with much greater conservatism.[21] In the external operation, either the route in front of or posterior to the sternomastoid may be taken, as advocated by various authors. We feel by the internal route the prone position with the use of suction is to be preferred. No general anesthetic is used. In the adult, topical applications of 10 per cent cocaine or 2 per cent pontocaine may be helpful, but in the child nothing is needed. The child is swaddled and held in a position similar to that used in a tracheotomy operation, mouth gag in place, tongue lifted forward gently. Feeling the abscess with the index finger of the left hand, a guarded knife blade is inserted cutting edge upward. A quick cut is made and suction used immediately. Then the opening is spread by means of a closed hemostat inserted into the wound. The hemostat is opened, spreading the structures, and suction is used to empty the pharynx. No packing is used; but the cavity is swabbed before removing the mouth gag. Spreading of the wound may be necessary from day to day until the cavity heals. General supportive and tonic treatment is indicated in all cases.

In the early or pre-abscess formation stages, heat to the neck, moist compresses, warm or hot gargles to promote hyperemia and short wave diathermy treatments are beneficial, and ease the patient. Supportive measures, tonics, feeding, forcing fluids, intravenous saline and glucose, if swallowing is too painful, should be instituted. Proper local treatments to the nose and throat and the use of suction if accumulated mucus cannot be gotten rid of. Incision and scarification of the inflamed and involved part for depletion is advocated by some, even while it is known that pus has not developed. But this is condemned and considered unwise by others due to the possibility of opening up channels for infection.

TABLE I

ADMISSION DIAGNOSES

1. Laryngeal obstruction, syphilis.
2. Retropharyngeal abscess, congenital syphilis.
3. Abscess and edema of the larynx; retropharyngeal abscess.
4. Cardiac dilatation.
5. Peritonsillar abscess. Abscess of the pharynx?
6. Abscess of the pharynx, laryngeal edema.
7. Retropharyngeal abscess. Four cases.
8. Swelling of neck; difficult nursing; dyspnea.
9. Hypertrophied and obstructive tonsils and adenoids.
10. Retropharyngeal abscess. Cervical adenitis. Suppurative otitis media.
11. Ruptured retropharyngeal abscess. Suppurative otitis media, left.
12. Hyperpharyngeal bleeding.
13. Dyspnea, fussy for three weeks.
14. Dysphagia, swelling of neck.
15. Dyspnea.
16. Sore throat, fever, glands of neck.
17. Dyspnea, laryngismus stridulus.

TABLE II

SYMPTOMS COMPLAINED OF

1. Difficult breathing two days. Difficult swallowing. Painful throat.
2. Sore throat, stiffness of neck. Difficulty in breathing and swallowing.
3. Sore throat, painful swallowing, restlessness.
4. Left side of neck sore and tender, hoarseness, painful swallowing.
5. Pain in right side of throat and neck. Painful swallowing.
6. Fever, painful swallowing, sore throat, stiff neck.
7. Pain and swelling of neck. Pain on swallowing.
8. Difficult breathing, marked. (Dyspnea, obstructive.)
9. Difficult breathing. Inability to nurse. Swelling left side of neck.
10. Sore throat, impaired breathing.
11. Sore throat with swelling of right side of neck.
12. Painful opening of mouth, painful swallowing, swelling of glands of neck.
13. Sore throat, fever, pain on swallowing.
14. Abscess of right ear canal, discharging, swelling of right side of neck. Painful swallowing.
15. Discharging, bleeding left ear, spitting of blood; vomiting of blood, painful swallowing.
16. Fretful and fussy with fever for three weeks. Some difficulty in breathing.
17. Swelling of neck and painful swallowing.
18. Some impairment in breathing.
19. Sore throat, swelling of neck and fever.
20. Marked difficulty in breathing. Restlessness, cannot sleep.

TABLE III

OPERATIONS

OPERATION	NO. OF CASES
None	2
Postmortem pharyngoscopy and laryngoscopy	1
Tracheotomy for impending suffocation	3
Internal, followed by external opening	3
Ligation of common carotid artery	1
Tonsillectomy and adenoidectomy followed by intra-oral opening	1
Incision in hypopharynx with punctures into edematous arytenoids	1
Intra-oral incision and drainage	11
Transfusions	4

TABLE IV

COMPLICATIONS

	NO. OF CASES
1. Hemorrhage common carotid and vertebral artery	1
2. Hemorrhage, probably jugular vein	1
3. Otitis media, suppurative	2
4. Asphyxia	2
5. Rupture of abscess into trachea	2
6. Edema of larynx	2
7. Enlarged Thymus	1
8. Abscess and necrosis of cartilage of larynx	1
9. Miliary tuberculosis	1
10. Vertebral destruction	1
11. Syphilis	2
12. Cardiac dilatation	1

CONCLUSION

The conclusions I have personally reached in the study of this condition are:

1. The involvement in adults is not nearly so uncommon as the literature would lead us to believe.

2. There seems to be a paucity of cases between the ages of ten and twenty-five years. The retropharyngeal lymph glands atrophy about the age of ten years.

3. That the involvement in adult life is due to secondary or hematogenous transplant.

4. That hemorrhage complicating retropharyngeal abscess is a most grave symptom and calls for immediate ligation. Procrastination spells disaster.

5. Tuberculous retropharyngeal abscess in the adult need not be dependent upon cervical vertebral involvement.

60 MEETING STREET.

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XXXIV

PAGET'S DISEASE, PITUITARY TUMOR AND ABSCESS OF THE SPHENOID SINUS: REPORT OF A CASE

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Paget's disease was (until the advent of the roentgen-ray) considered to be a rare disease. Now that physical examinations are more complete it has been more often recognized, but even yet it is one of infrequent occurrence.

There are phases of the pituitary syndrome so common that we usually keep them in mind when making a diagnosis. However, neoplasm of the gland is not common, and even when present it is not usually accompanied by the symptoms of distributed pituitary function.

Purulent infection of the sphenoid sinus is relatively common, but when Paget's disease, pituitary tumor and sphenoiditis are found in one patient, it is to say the least, unusual. In such a case, the symptoms, the findings and the clinical interpretation are difficult to untangle.

PAGET'S DISEASE

This is primarily a disease of the bones. If there are other structures involved in the process, no definite proof has been forthcoming. The bones most commonly affected are the skull, the vertebrae and the bones of the leg, but any or all bones may be involved.

The causative factor has not been determined. Some think it is a chronic infection of the bones but the evidence is not convincing. Discovery has definitely been made that there is a disturbance of metabolism and this particularly of calcium, since the osseous tissues are so fundamentally involved. In this process there is a retention of calcium, phosphorus and magnesium and a loss of sulphur. The calcium content in the blood is generally within normal limits, but there are cases reported in which the blood calcium has been higher than normal.

An endocrine dyscrasia as an etiologic factor has been investigated from many angles and there is considerable evidence that an imbalance in the secretion of hormones starts decalcification of the

bony tissues. The parathyroids in particular have been the subject of extensive research since they are known to control calcium metabolism. In some cases of Paget's disease actual tumors of the parathyroids have been discovered but the relationship has not been proven. Berman thinks that the adrenal cortex is closely associated with calcium demobilization; also that there is a cortical hormone which assists the bones to take calcium from the blood. Morlig and Murphy[1] feel sure that Paget's disease is associated with diabetes and therefore, consider the metabolism of carbohydrates as a cause. Dietary indiscretions, too much starch and too little protein, have all been investigated in a study of the etiology but have been found wanting. Since the anterior lobe of the pituitary has to do with skeletal growth and development may we not suspect that a disturbance here has to do with osteitis deformans. Probably at some future time the endocrines will give up their secret and we will know what chemical changes take place to bring this disease into being.

The disease comes on so insidiously that the patient scarcely knows there is anything the matter with him until deformities of the bones begin to take place. Pain is the most prominent and usually the earliest subjective symptom. It may be of all grades and varies from a dull soreness to the most intense migraine. Headache is often present and is usually worse at night or in the early evening. The head enlarges (Fig. 1), the bones that bear weight bend and the patient actually becomes shorter in height. In the advanced stages, all the bones in the body may be involved, even those of the face. The bones of special structure are not spared and the petrous portion of the temporal bone frequently is affected, so much so that the osseous changes cause disturbances of the hearing mechanism and deafness results. Anson and Wilson,[10] in a discussion of the temporal bone changes in osteitis deformans, say: "The petrous portion of the temporal bone possesses osseous tissue of an unusual type; this means that the progress of the disease in the temporal bone may be different from that in other skeletal parts and that its spread may do more than weaken long bones and vertebrae, actually causing a loss of hearing or a disturbance of the sense of balance."

Lindsay and Pearlman,[7] writing on Paget's disease and deafness, say that the most common characteristics of the deafness in these cases appears to be an impairment of the inner ear. The same thing probably happens to the optic foramen in those cases where there is a primary optic atrophy. It may be that the small retinal hemorrhages which often occur in this disease are due to softening and distortion of the bony optic canal.

Fig. 1. A-P view of skull; note thickening of skull and the rough, irregular surfaces.

The gross pathology of the bones shows them to be soft and easily cut with a knife. It is at this stage that deformities occur, namely: bowing of the long bones, enlargement of the skull, etc. Later the bones become hard and of increased thickness. There is a deposit of subperiosteal bone on the long bones and the skull. The surfaces are rough and irregular. This thickness is most strikingly seen on the cut surface of the skull cap and a pathologic diagnosis can be made from this alone.

The microscopic pathology shows first a replacement of original bone by connective tissue and then a substitution of finely porous cancellous bone which gradually becomes harder (Fig. 3). Absorption and ossification go on together, but the latter outstrips the former, so that the bone becomes thick though still finely porous. The lamellar systems are irregular. This is seen in no other disease

of bone. New and old bone are separated by ground substance of connective tissue. The medullary cavity is filled with connective tissue.

PITUITARY

The pituitary body consists of an anterior and a posterior lobe. The anterior lobe is epithelial in structure and is derived from Rathke's pouch, an upgrowth from the pharynx. Epithelial rests may remain from which tumors arise known as cranio-pharyngiomas. The posterior lobe of nervous structure is developed from the floor of the third ventricle. It consists of a pars nervosa and a pars intermedia. Nonmedulated nerve fibers connect the posterior lobe with the hypothalamus. The pars intermedia is an epithelial investment of the pars nervosa, also derived from Rathke's pouch. It is composed of basophil cells. The active hormone of the posterior lobe is derived from the cells of the pars intermedia.

The anterior lobe is made up of three types of cells. (1) Acidophils, the granules of which stain red with eosin. (2) Basophils, whose granules stain with hemotoxylin. The eosinophil and the basophil cells are classified together as chromophil cells, and (3) chromophobe cells with nongranular cytoplasm which stains faintly and makes up the greater part of this lobe.

The anterior lobe governs connective tissue growth, in particular that of the bones and mental development of the reproductive organs. The posterior lobe yields an extract of remarkable potency. The crude product is known as pituitrin. It contains several hormones with different actions, of which the most important are the vasoconstrictor, the oxytoxic and the antidiuretic.

Hyperpituitarism is characterized by overactivity of the gland and such symptoms as gigantism or acromegalia may arise. If there is underactivity of the gland, or hypopituitarism, there will be dystrophy of the genitals and depression of the sexual function. Adiposity may be marked with deposits of fat in the breasts, hips, buttocks and lower abdomen.

If the function of the gland is interfered with by an abnormal growth we may have the symptoms of overactivity, or the symptoms of underactivity, or we may have none of these symptoms at all. Our attention is attracted only by the signs of intracranial pressure, such as disturbed vision, headache, choked disc, etc. The x-ray findings are usually present with an enlarged sella. Engelbach states that tumor constitutes a part of the pituitary syndrome in only about 4 per cent of the clinical cases.

Fig. 2. Lateral view of skull; note destruction of sella and roof of sphenoid sinus. Small remnant of posterior clinoid process is yet present.

Tumors of the pituitary gland are adenomas. They are divided into three types: (1) Chromophobe; (2) acidophil, and (3) basophil.

The chromophobe tumor is much the commonest form. The cells are nongranular so that they appear clear and show a very characteristic alveolar grouping, the groups being separated by fibrous septa. This type of tumor can usually be recognized by the alveolar arrangement without having recourse to special study of the granules. The symptoms are usually those of pituitary insufficiency and similar to the changes produced in an animal when the anterior lobe has been removed. The function of the cells is not known and their overgrowth gives no positive symptoms. The negative symptoms of insufficiency are the result of compression of the more actively functioning eosinophil and basophil cells. The adenoma, at times, remains confined to the sella, causing interference with skeletal growth and sexual development. The acidophil adenoma is much less common and the basophil the rarest form of pituitary tumor.

Malignant adenoma of the pituitary is a rare tumor. In Cushing's series, there were only three malignant tumors compared with 159 innocent ones.[13] The tumor destroys the base of the skull. It bursts through the roof of the sella and invades the floor of the third ventricle. The cells which are of the chromophobe type are grouped in irregular masses.

Deformity or erosion of the sella turcica is the one sign constantly suspected in any syndrome which may be referable to the pineal gland. A diagnosis of neoplasm of the hypophysis depends not only on unquestionable enlargement of the sella, but also on local pressure signs. In the majority of cases there will appear at some time a deformity of this bony structure. Bitemporal heminopsia may or may not be present. Visual field changes are by no means typical and they are never alike in the two eyes, usually one eye being well in advance of the other. If chiasmal interference coming from below, presses on these nerve structures, field deformities will result, but if the pressure is further back or is directed downward, there may be no field findings. The perimeter defects resulting from hypophyseal neoplasms vary so much from the classical type that they are more often attributed to some such benign condition as hysterical blindness, toxic amblyopia, or secondary infection from focal diseases in the sinuses and teeth rather than to a localized pressure upon a limited number of fibers of the optic chiasm or tract. Pituitary neoplasm may produce a transient fleeting blindness, and this is the most constant ocular symptom due to these tumors. When growth is directly anterior and presses on the chiasmic fibers, exactly at their crossing, classical hemianopsia in the temporal fields results. As the process goes on, the tumor may not infrequently decompress itself either through the posterior wall of the sphenoid or by eroding the posterior clinoid process. Choked discs, papilledema and optic atrophy are at times present, even in the earlier stages, but more often these are terminal findings and come on from intracranial pressure due to the large size of the neoplasm. Primary optic atrophy may be found here as well as in Paget's disease. These symptoms and nasopharyngeal signs (decompression of the tumor into the pharynx) of rhinorrhea develop late and should not be anticipated. Headache is the most common sign of general intracranial pressure. It may, in exceptional cases, be localizing in type. The headache is often on the order of migraine occurring over one eye or deep in the orbit. It is fairly constant, seldom is there a remission and the lower part of the cranium is more often the seat of pain, or it may be deep in the midorbital region. It also radiates to both temples and to the

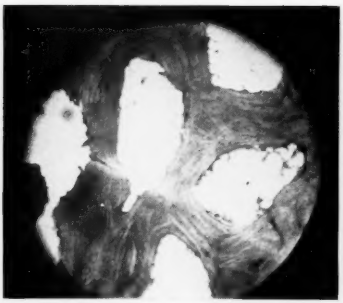

Fig. 3. Bone changes; note thickened trabeculae; also mosaic structure of new bone which has replaced old bone.

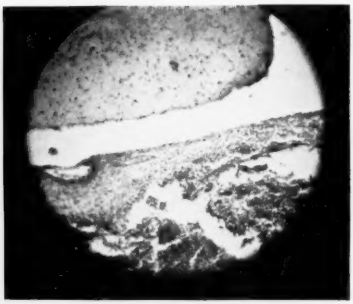

Fig. 4. Above, normal parenchymatous brain tissue; below, thick infiltrated meninges overlying brain.

Fig. 5. Lining of sphenoid sinus; thick infiltrated substructure and irregular epithelium.

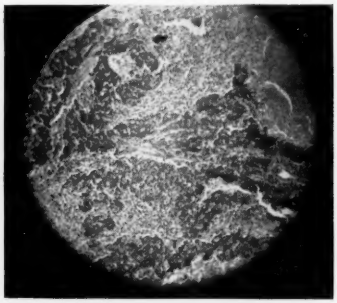

Fig. 6. Pituitary tumor; note tendency to formation of alveolar structure and tendency to invasion.

vertex and the postoccipital region. Yet to be of any great value as a diagnostic sign, other symptoms of pituitary disease should be present, particularly hypoactivity of the gland. These headaches are not unlike those produced by the sphenoid sinus. Here, the pain is in the occipital area at the base of the brain or at times mid-orbital and fairly constant. However, there are always remissions if headache is due to an infection in the sinus. It is often unilateral and of the migraine type. In sphenoiditis, it is not uncommon for pain to be referred to the ear, to the mastoid process and to the eye-ball itself. With a positive x-ray film of the sphenoid sinus and a purulent postnasal discharge, one would need to rule out the sphenoid as a cause of persistent headaches.

REPORT OF A CASE

A white man, aged 62, occupation stock grower, was first seen on August 21, 1936. He complained of severe headache. This he described as being constant other than in sleeping and even then he would frequently be awakened by the pain in his head. It was deep behind the eyes, dull, boring in character and radiated to the occiput, to the base of the skull and, at times, over the left eye. He was hard of hearing and was also troubled with a pharyngeal discharge and an obstruction to nasal breathing, particularly in the right nostril. He was cross and irritable, his memory was poor and he could not concentrate.

He was a well developed man who looked older than his years. His gait was deliberate and he was slightly stooped. His expression was that of one suffering pain. His systolic pressure ranged from 170 to 200 and the urine and blood Wassermann were negative. The nasal mucous membrane was red and dry. The nasal septum was so deflected that the left nostril could not be examined. The tonsils appeared normal and there was a mucopurulent discharge in the pharynx. The hearing was reduced in both ears and the drums were retracted. He heard the conversational voice with little effort. An x-ray of the sinuses showed some blurring of the ethmoids and sphenoids. The eyes were normal except for some signs of arteriosclerosis in the fundus. The refraction was normal and the vision 20/30 in each eye, which was corrected to 20/20 with 50D sphere. I made a diagnosis of a mild infection in the ethmoids and sphenoids, after which I advised him to have the nasal septum resected, since no treatment could be applied until this was done. To this both he and his family objected.

I did not see him again until December 6th, 1937, or fourteen months after his first visit. During this interim he had had no relief from the headache, in fact it had grown more intense. From the time I had seen him last, until this visit he had run the gauntlet of quacks and charlatans. The tonsils had been removed and finally he had been referred to a very competent neurosurgeon who, after a prolonged examination sent him back with a diagnosis of Paget's disease. The bony changes were limited to the skull; therefore, he was treated for Paget's disease but without relief. While the bony changes were limited to the skull, this did not account for the severe constant headache, and, hence, he had returned seeking relief from the pain.

He was immediately sent to the hospital where an examination showed: (1) Some pallor of the discs; otherwise the fundus was normal. The visual fields were normal and his vision was 20/50 in each eye. (2) Erythrocytes 5,450,000; lymphocytes 26; monocytes 1; polyps 72; basophiles 1. (3) Urine normal. (4) Cerebrospinal fluid contained twelve cells and the Pandy test was slightly positive. (5) A complete roentgen study of the bones showed evidence of osteitis deformans in the skull and a beginning of the same disease in the femurs and humeri. A lateral view of the skull showed the sella turcica badly eroded and in places gone. The clinoid processes, both anterior and posterior, were destroyed and there was no shadow cast of the posterior wall of the sphenoid (Fig. 2). At this examination all the sinuses, other than the frontals, were opaque.

Though he was extremely ill and symptoms of impending meningitis approaching, I resected the septum, thinking probably I could clean away the debris and possibly promote drainage by decompressing the pituitary area; however, this was not attempted because of the extensive pathology and the poor condition of the patient. For several days he felt better, but on the third, fourth and eighth postoperative days, his temperature rose to 103 degrees F., accompanied by symptoms of meningeal irritation, then returned to normal. His cerebrospinal fluid now showed 160 cells and was under considerable pressure. The white blood cells were now 12,800 and the segmented cells had increased to 80 per cent.

From this date his condition grew worse, and on December 27 he was stricken suddenly with terrific pain in the head, followed by a profound coma. His temperature rose to 107 degrees and he died with all the symptoms of acute fulminating leptomeningitis.

Autopsy: A thick skull cap was removed. In some places it was a full centimeter thick. The dura was covered with pus over the left frontal lobe (Fig. 4). This extended down to the base, where it ended in a pool of pus and covered most of the base of the brain. The pituitary gland was lying in a necrotic mass (Fig. 5), which when elevated out of its bed, brought with it fragments of bone, i. e., pieces of the sella, the clinoid processes and bony fragments of the posterior wall of the sphenoids. The sphenoid sinuses could be viewed from above. The dura was now lifted and the entire region above the sella, sphenoid sinus and hypothalamus was one infected mass. The pituitary gland was large, soft and indented with necrotic areas. It measured about 35 mm. in diameter. The pia arachnoid and the subarachnoid spaces were infiltrated with infection. The antrums and ethmoids were filled with pus.

Pathologist's Report: (1) Rather thick section of skull. (2) Brain with streaks of pus following the subpial blood vessels. When cut, the brain showed no parenchymatous lesions but there is thick pus in the lateral ventricles and in the walls are numerous punctate hemorrhages. (3) Pituitary about 35 mm. in diameter with ragged borders.

Microscopic: (a) Brain shows subpial collection of pus. (b) Pituitary shows areas of necrosis and areas of medullary type of carcinoma (Fig. 6). (c) Bone of skull shows greatly thickened fibrous trabeculae characteristic of Paget's Disease.

SUMMARY

1. Paget's disease is a disease of the bones, while the pituitary governs the growth and development of them.

2. Both Paget's disease and pituitary neoplasms give rise to headache of a similar nature and both may cause enlargement of the skull.

3. The subjective eye symptoms are similar in both diseases and these alone may first bring the patient to the physician.

4. The headache caused by an infection of the sphenoid sinus or by neoplasm of the pituitary and at times the headache of Paget's disease may be confused with either of the above.

5. In my own case there were no symptoms or findings of visual disturbance and there were no signs of hypo- or hyperactivity of the pituitary gland.

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XXXV

MICROSCOPIC CHANGES IN OSTEOMYELITIS OF THE FRONTAL BONE*

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This presentation is a review of certain laboratory specimens of osteomyelitis of the frontal bone to determine if possible the order of the cellular changes in the bone and bone marrow as the result of infection. It was pointed out some time ago that in cases of osteomyelitis of the frontal bone fibrosis occurred very quickly, and the question arose whether this was a defensive reaction and what, if any, was its surgical importance. That study was confined to the marrow changes in the disease. Many of our findings have been known, or suspected to be true and seem to justify the radical removal of the frontal bone in osteomyelitis.

The specimens obtained from the surgical amphitheater were subjected to x-ray, photographed, then cut into suitable blocks according to prearranged diagrams, so that the microscopic pathology could be correlated with the clinical and surgical findings. The complexity and normal variability of bone marrow makes discussion without consideration of the related anatomy and physiology difficult. When the marrow has been altered by infection, the bacteriology and immunology are further considerations.

In the normal frontal bone, the microscope reveals fat or yellow marrow, and red or blood marrow, the distinction being one of degree in activity. The former is a state of hypoactivity, the latter of active hemopoiesis (blood cell production). Red marrow consists of the ancestors of the blood cells. Innumerable variants of immature cells crowd the microscopic field. The erythrocytes are believed to be produced from the central portion of the marrow where the oxygen tension is low. The cells from which they are derived, either endothelial or connective tissue cells, are still a point of controversy.

The granulocytes find their genesis at the periphery of the marrow mass where the oxygen tension is higher. These are gen-

*This work was done in the Mosher Laboratory of the Massachusetts Eye and Ear Infirmary.

erally considered to arise from connective tissue. The belief that the expulsion of nuclei from the normoblasts and the resultant liberation of nucleic acid stimulates granulopoiesis (white cell production) is based on experimentation.

The circulation in bone marrow is of the closed type. It is sinusoidal and sluggish, and the capillaries from the nutrient vessels leave at right angles. At any one time a large part of the sinusoids are closed or collapsed and erythropoetic (red cell producing) in function.

The theory of rhythmic opening and closing of the minute capillary channels to admit the immature blood cells to the circulation has some foundation.

In the marrow the ratio between red and white cells is that of one to one. In the blood the ratio of red to white cells is that of 700 to 1. This is explained by the relatively longer life of the erythrocyte. When bone marrow is depleted by infection, intoxication, or starvation, the erythropoetic function is always the last to disappear. When recovery ensues, it is the first to become active.

To explain and analyze the various activities, functions and transformations of bone marrow, the role of endocrines, and enzymes must be correlated. A study of bone marrow lucidly written and concisely presented by Sabin[1] was the source of much of the above information.

Mosher[2, 3] has demonstrated that infection from the frontal sinus spreads to the diploe, dura, and brain by thrombophlebitis of the perforant veins and by direct extension through the eroded Haversian canals. The diploic veins draining the sinus and surrounding bone via the perforant veins traverse the diploe for a short distance, then go to the dura, then to the sagittal sinus and frontal lobes of the brain.

In osteomyelitis of the frontal bone certain changes occur in a definite pattern. At first the normal hypoactive marrow is simple in composition and apparently made up entirely of fat cells. These are packed so closely together that they crowd the essential marrow elements into stellate or angular masses (Fig. 1).

Within the confluent angles of this network are hematopoietic vascular channels which in the quiescent state are collapsed and hard to see. In the active stages these channels become visibly engorged. This engorgement is the first change seen in hypoactive marrow as a response to infection. The channels dilate readily and give a false impression of hemorrhage (Fig. 2).

Along with the hyperemia, increased activity is found within stellate masses. Hyperplasia follows and the marrow cells crowd out and obliterate the fat cells. The manufacture of white cells is more prominent than red cell production. The vascular congestion is nearly overshadowed by the tremendous overgrowth of the essential marrow cells (Fig. 3). At this stage it is possible for this activity to subside and the marrow to revert to its original state of hypoactivity.

With the advent of infection the vessels and sinusoids become thrombosed; they fill with fibrin and purulent debris, at first resembling a log-jammed stream. Whenever there is purulent accumulation or inflammatory exudate, osteoclasts are seen absorbing the marrow partitions which become wafer thin. The osteoclasts appear to be making space to relieve the pressure caused by the accumulation of debris (Figs. 4 and 5).

Polymorphonuclear leucocytes are found in quantity only in the thrombosed vessels and pus pockets. These often seem to be walled off by fibrosis. Along the dural surface irregular spicules of new bone are being formed by the osteoblasts in an apparent effort to reapproximate the endosteum and inner table which have been separated by the dissecting exudate and edema. The tissue elements seem to be working to bring about isolation and segregation of the invading infection (Fig. 6).

Around the infected areas fibrosis takes place. A fibrous ring begins to form along the walls of the marrow spaces. This fibrous band gradually engulfs and destroys the marrow elements (Fig. 7).

In the wake of the advancing fibrous tissue, osteoblasts are seen laying down new bone. Osteogenic buds arise from the bone marrow partitions. These buds extend to form trabeculae bridging the gaps in the partitions and filling in the marrow spaces. The infected area becomes surrounded by fibrosis and a wall of gnarled bone which resembles the burls or rosettes in the bowl of a good briar pipe. The fibrosis and osteogenesis erect a barrier to the infection which is diverted toward the dura (Figs. 8 and 10).

Macmillan[4] has pointed out that the x-rays of chronic frontal sinusitis show a marked increased density of the bone surrounding the sinus. This can be interpreted as a wall of resistance built up as the result of a previous infection, and presumably decreases the probability of subsequent osteomyelitis of the frontal bone (Fig. 9).

SUMMARY

1. The microscopic picture in acute fulminating osteomyelitis of the frontal bone justifies radical operative procedures. Since the use of radical operation in the treatment of osteomyelitis of the frontal bone, an estimated decrease of 70 to 80 per cent in mortality has been noted.

2. The unaided eye cannot detect the limitations or the spread of infection.

3. The reaction of bone marrow can be followed through definite sequences by histological examination.

4. Fibrosis occurs early, and indicates that infection may be shunted toward the dura.

5. Fibrosis does not preclude the possibility of serious complications.

6. Like osteomyelitis in other bones, the reaction of bone marrow in the frontal bone is to encapsulate the infection. Unlike osteomyelitis in other bones, osteomyelitis of the frontal bone is potentially more deadly because of its proximity to the dura and the frontal lobes of the brain.

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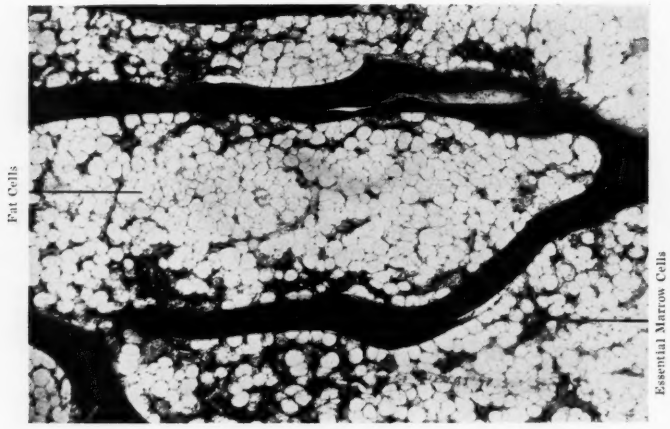

Fig. 1. Photomicrograph of hypoactive bone marrow. The fat cells crowd the essential marrow cells into stellate or angular masses.

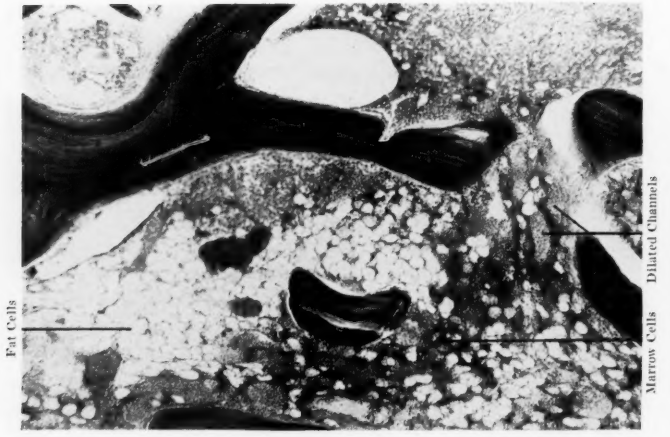

Fig. 2. Photomicrograph showing the early stages of marrow activity. The dilated vascular channels give a false impression of hemorrhage.

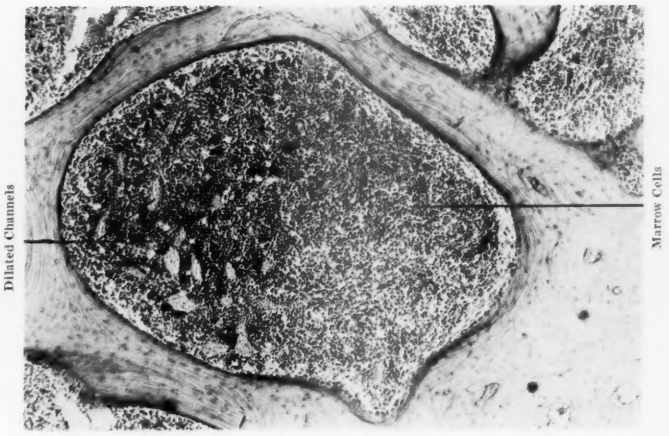

Fig. 3. Hyperplasia. The marrow cells have crowded out and obliterated the fat cells. The vascular congestion is nearly overshadowed by the tremendous overgrowth of the essential marrow cells.

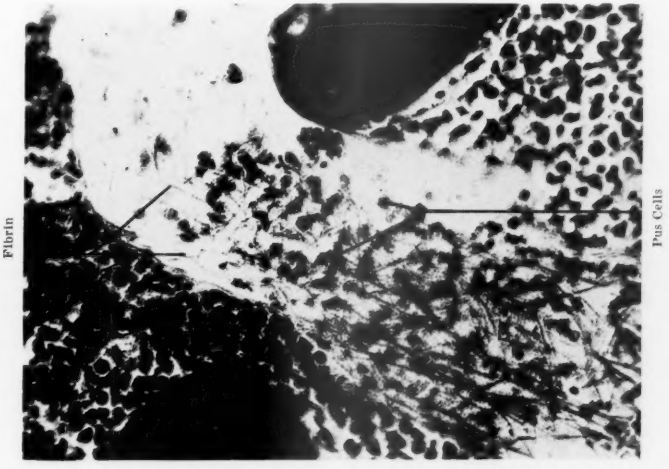

Fig. 4. Thrombosis. Fibrin and the enmeshed purulent debris resemble a log-jammed stream.

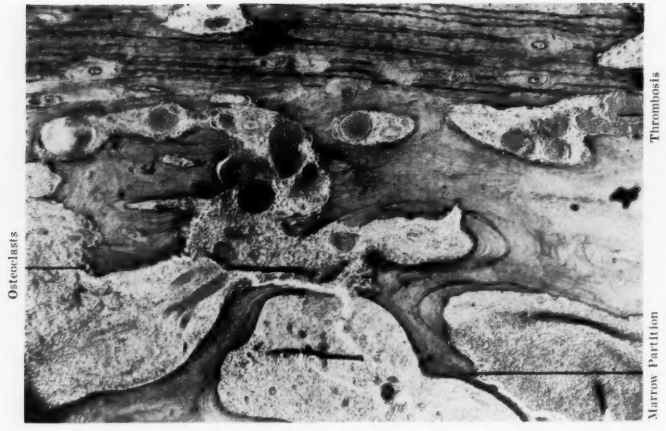

Fig. 5. Thrombosis and purulent accumulation. The activity of the osteoclasts is noted by the ragged appearance of the bone partitions.

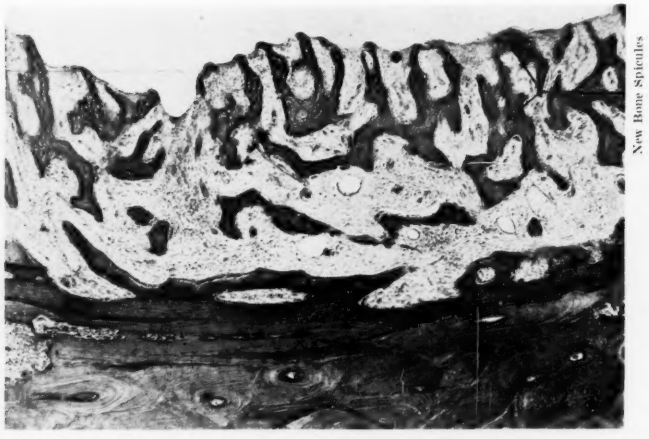

Fig. 6. Photomicrograph showing irregular spicules of new bone along the dural surface of the frontal bone.

Fig. 7. A band of fibrosis formed around the periphery of the marrow elements. Note osteogenic buds beginning to develop along the marrow partitions.

Fig. 8. New bone trabeculae arise from the bone marrow partitions filling in the marrow spaces.

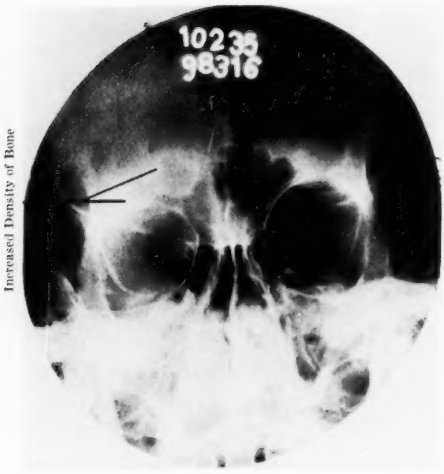

Fig. 9. Photograph of x-ray showing chronic right frontal sinusitis. Note the marked increased density of the bone surrounding the sinus.

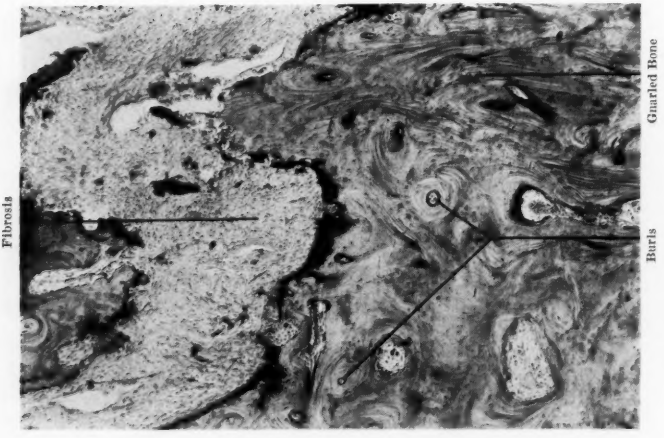

Fig. 10. Photomicrograph showing fibrosis and a wall of gnarled bone around the infected area. Note the burls or rosettes of the surrounding bone.

XXXVI

BACTERIOLOGICAL STUDIES OF ACUTE INFECTIONS OF THE MIDDLE EAR

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The type of bacteria in acute infections is becoming more and more important with the advent of many specific therapeutic agents which are themselves gaining considerable popularity. The bacteriological diagnosis is just as important in acute infections of the middle ear as in pneumonia. These cultures are very easily made from the myringotomy knife at the time of the operation by placing the knife immediately into a sterile tube, care being taken not to touch the point of the knife to anything that will wipe it clean or contaminate it. In this way the laboratory then obtains an uncontaminated culture to work with. In this study cultures were made only aerobically on blood media directly from the knife in cases where myringotomy was done, or by sterile cotton applicator in cases with spontaneous rupture. Anaerobic cultures were not done. There was a fairly large number (23.7%) of the cases cultured from the myringotomy knife which showed no growth. If on a subsequent visit, there was any discharge, a second culture was done.

The most extensive study of the bacteriology of acute infections of the middle ear and mastoid was done by Page,[1, 2] who reported a series of 836 cases done in one year in the Manhattan Eye, Ear and Throat Hospital during 1934. He found several striking things, namely:

1. He isolated 15 different types of bacteria.

2. Mastoidectomy was necessary in three types of infections only; the hemolytic streptococcus, pneumococcus Type III, and the staphylococcus.

3. By far the greatest number of cases fell into three groups; 30 per cent streptococcus hemolyticus, 23 per cent staphylococcus and one per cent pneumococcus.

4. There was no growth obtained in 22 per cent of his cultures.

5. Mastoidectomies were done on 5.1 per cent of his cases. There were three times as many mastoids in those cases where the drum ruptured spontaneously as compared to those who had an early myringotomy.

6. There is considerable seasonal variation; 36 per cent in winter, 40 per cent in early spring, 13 per cent in summer and 10 per cent in fall.

Page suggests that the type of infection if cultured at the time the myringotomy is made, may indicate some prognostic value in view of the fact that mastoidectomy was necessary in only three types of infections.

Hadjopoulos[3] reported 91 "selected" cases on which he made an extensive bacterial differential study. His principal finding was that "of the various pneumococci found, Type III is the one invariably encountered in the middle ear and mastoid infections." He gives a particularly bad prognosis to this group of cases.

In this series we are reporting the results of a bacteriological study in 468 acute cases. There were 243 males and 225 females. The youngest was three months and the oldest was 63 years of age. All cases were considered acute infections, no chronic draining ears are included in this series. The average duration of the illness before we saw the case was eight days, in no case was there a history of illness longer than seventeen days. Of these 468 cases, 365 were seen before the tympanic membrane had ruptured and a myringotomy was done; 103 had ruptured spontaneously and were draining when we first saw the case. There are eleven patients reported in this series who had two different middle ear infections. The first had apparently cleared, as manifest by a canal free of pus and a healed membrane, and the patient appeared clinically well when the second infection developed and a second myringotomy was done or spontaneous rupture occurred.

In this series of cases 136 showed no growth on first culture; six of these were among those in which the membrane had ruptured spontaneously. These six seemed to be sterile inasmuch as they showed no growth on subsequent cultures or were dried up when seen on the next visit. In a few of the 136 cases that showed no growth from the myringotomy knife culture, running ears developed and when seen on a second visit, if this was the case, the ears were recultured. In twenty of these cases (15%) organisms were

demonstrated on this reculture while 116 remained apparently sterile. Because of this fact the total number of cultures reported are 489 on 468 cases.

In the 353 cultures showing growth there were thirteen types of organisms isolated (Chart I). The gram positive coccus was incriminated in 337 (95.6%) cultures, including streptococcus hemolyticus, streptococcus viridans, staphylococcus and pneumococcus (Chart II). These were not always pure cultures, as sometimes more than one coccus type was found in a given culture, and in a few cases the coccus was found in combination with the gram positive or gram negative bacillus. In only sixteen cultures (4.5%) gram positive cocci were not found and the infection was charged to the bacillary group of organisms.

CHART I

Streptococcus Hemolyticus	B. Influenzae
Streptococcus Viridans	B. Pyocyaneous
Staphylococcus	B. Diphtheroid
Pneumococcus	B. Alkaligenes
B. Diphtheriae	Friedlander's Bacillus
B. Proteus	M. Flavus
B. Coli	

The streptococcus hemolyticus and the staphylococcus were the worst offenders. The hemolytic streptococcus was found in pure culture in ninety (25%) cultures and in combination with some other organism, not the staphylococcus, in four cultures. The staphylococcus was found in pure culture in 113 (32%) cultures, and in combination with other organisms exclusive of the hemolytic streptococcus in thirty-three cultures, making a total of 41 per cent. The hemolytic streptococcus and the staphylococcus were found jointly in forty-two cultures (11%) and these two organisms with a streptococcus viridans in one case and with a pneumococcus Type II in one case. The pneumococcus* was found alone in forty-two cultures (11%), distributed among the various types as follows: Type I in ten cultures, Type II in fifteen cultures, Type III in one culture, and Type IV in sixteen cultures. The pneumococcus was also found in combination with the staphylococcus, the hemolytic streptococcus, B diphtheria, and streptococcus viridans, in all in twenty cases. The streptococcus viridans was found in seven cases

*The identity of the different types of the pneumococcus was not carried beyond types I, II, III and IV for practical reasons.

only in pure cases and in combination with some other organism in fourteen cultures. The rest of the cultures were divided one and two each among the remaining types of bacteria.

In this whole series of 468 cases only thirty-one (6.6%) went on to develop surgical mastoiditis. In this group nineteen or 5.2 per cent were in that group that had had myringotomies, and twelve (11.6%) were in ears that had ruptured spontaneously (Chart III). Of these thirty-one cases, fifteen (48%) were due to pure culture of streptococcus hemolyticus, six (19%) were due to the staphylococcus, three to pneumococcus Type I, and one to Type IV, three to a combination of the hemolytic streptococcus and staphylococcus combined, one to staphylococcus and pneumococcus Type IV, one to staphylococcus and B. proteus combined, and one to the Friedlander's bacillus and one** showed no growth. All these patients recovered.

CONCLUSIONS

It seems significant that 116 cultures (23.7%) showed no growth even though clinically these patients had an acute otitis media. Page[2] reported that cultures from 23 per cent of his cases showed no growth and he seemed to regard it as, "Probably being in a faulty technique." We cannot agree with this viewpoint, for we feel there must be some other factors, otherwise why should these ears clear as soon as the pressure is released by myringotomy, even without much drainage through the external canal. In six of our cases there was enough pressure to produce spontaneous rupture of the membrane. The character of the discharge in these cases was usually mucoid instead of purulent in character.

The gram positive cocci accounted for the greatest percentage of the infections in this series (95.6%). However, of this group the staphylococcus was found in 32 per cent of the cultures. If we add these to the ones which showed no growth we have a total of 229 cultures or 47 per cent. Sulfanilamide and sulfapyridine are used extensively today in chemotherapy of certain types of infections. Many are giving these drugs empirically, and yet we know they are not without their toxic effects on the patient, sometimes seriously. In this series there are 47 per cent of the cases that would have received little or no benefit from these drugs, had we used them without determining the type of infection, and damage might

**The myringotomy cultures on this case showed no growth and the culture taken at the time of operation for mastoid was lost by accident.

have been done. There were 37 per cent streptococcus hemolyticus and 12 per cent pneumococcus, or a total of 49 per cent of these cases that we could expect benefit from the use of these drugs. There is an additional 4.5 per cent of the cases in which the bacteriological findings proved them to be non-coccal in type. The benefit to be derived from this particular chemotherapy in this group of infections is still doubtful, in view of our present knowledge.

There were thirty-one patients (6.6%) that went on to develop mastoiditis and were operated upon. Of the thirty-one patients, nineteen had had myringotomies and twelve a spontaneous rupture, but if one considers the number of myringotomy cases this is only 5 per cent of that group of cases, while 12 per cent of those that ruptured developed mastoids, or two and a half times as many. Page[2] reported three times as many mastoids among the cases which ruptured spontaneously as compared to those in which myringotomy was done.

In our series we did not find the large number of pneumococcus Type III infections that are reported by Hadjopoulos,[3] and among the pneumococcal infections we did have, there were no fatal cases. The duration of the illness was in some cases considerably longer.

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CHART II

ORGANISM ISOLATED	MYRINGOTOMY		RUPTURE SPONTANEOUS		TOTAL	
	No.	Pct.	No.	Pct.	No	Pct.
Streptococcus Hemolyticus	61	16	29	28	90	19
Streptococcus Viridans	5	1	2	2	7	2
Staphylococcus	82	23	31	30	113	24
Pneumococcus I	8	2	2	2	10	2
Pneumococcus II	13	4	2	2	15	4
Pneumococcus III			1	0.9	1	0.2
Pneumococcus IV	16	4			16	4
Staphylococcus and Streptococcus Hemolyticus	26	7	16	15	42	9
Staphylococcus and Streptococcus Viridans	4	1	5	5	9	2
Staphylococcus and Pneumococcus I	2	0.5			2	0.4
Staphylococcus and Pneumococcus II	8	2			8	2
Staphylococcus and Pneumococcus III	2	0.5			2	0.4
Staphylococcus and Pneumococcus IV	4	1	2	2	6	2
Staphylococcus, Pneumococcus II and Hemolytic Streptococcus	1	0.3			1	0.2
Staphylococcus, Pneumococcus II and B. Diphtheriae			1	1	1	0.2
Staphylococcus, Streptococcus Hemolyticus and Strep. Viridans	1	0.3			1	0.2
Staphylococcus and B. Diphtheriae	2	0.5	1	1	3	0.6
Staphylococcus and B. Proteus			1	1	1	0.2
Staphylococcus and Friedlander's Bacillus			1	1	1	0.2
B. Diphtheriae	6	2	2	2	8	2
B. Diphtheriae and Streptococcus Hemolyticus	4	1			4	1
B. Diphtheriae and Streptococcus Viridans	3	1			3	0.6
Friedlander's Bacillus			1	1	1	0.2
Friedlander's Bacillus, Streptococcus Viridans			1	1	1	0.2
B. Coli	1	0.3	2	2	3	0.6
B. Influenzia	1	0.3			1	0.2
B. Pyocyaneous	1	0.3			1	0.2
M. Flavus			1	1	1	0.2
B. Diphtheroid and B. Alkaligenes	1	0.3			1	0.2
No Growth	130	36	6	7	136	28
Total Number of Cultures	382		107		489	

CHART III

MASTOIDECTOMIES

ORGANISM FOUND	MYRINGOTOMY NO.	MYRINGOTOMY PCT.*	RUPTURE SPONTANEOUS NO.	RUPTURE SPONTANEOUS PCT.*	TOTAL NO.	TOTAL PCT.*
No Growth	1	0.8	—	—	1**	0.8
Streptococcus Hemolyticus	10	16	5	17	15	17
Staphylococcus	4	5	2	6	6	5
Pneumococcus Type I	1	13	1	50	2	20
Pneumococcus Type IV	1	6	—	—	1	6
Staphylococcus and Streptococcus Hemolyticus	1	4	2	21	3	7
Staphylococcus and Pneumococcus Type IV	1	25	—	—	1	20
Staphylococcus and B. Proteus	—	—	1	100	1	100
Friedlander's Bacillus	—	—	1	100	1	100
Total number of cases	19	—	12	—	31	

*These percentages represent the number of any one type of cases with this type of infection that developed mastoid.
**This was a myringotomy culture only. The culture taken at the time of operation was accidently lost.

XXXVII

GIANT FOLLICULAR HYPERTROPHY OF NASOPHARYNX: REPORT OF A CASE

FRANK J. NOVAK, JR., M.D.

CHICAGO

In 1925, Brill, Baehr and Rosenthal[1] called attention to a disease characterized by a generalized lymphadenopathy in which there occurs a characteristic hyperplasia of the lymph follicles without specific changes in the blood, and with or without enlargement of the spleen. The disease yields to x-ray therapy. In 1927, Symmers reported three similar cases, and Terplan in 1929 reported one. Since 1933, Symmers[2] has reported seven cases. The disease is regarded as a definite clinical entity, has been but recently recognized, and the number of cases reported is relatively small.

The changes in the lymphoid tissues are primarily, according to Symmers, "A numerical, and dimentional hyperplasia of the lymph follicles." In other words, there is an actual increase in the number of follicles, as well as an increase in the size of the individual follicles. Symmers further describes the cytologic details of the tissues as follows. The cells are (a) large hypochromatic embryonal cells, or shadow forms, and (b) hyperchromatic cells of the large lymphocytic type. Forms transitional between the two are discernible.

It is to be noted that the cases reported deal with the lymph nodes—cervical, inguinal, mesenteric, etc. As far as the writer knows, no case has been reported in which the hyperplasia has involved the terminal lymph nodules, such as the adenoid and the tonsil.

REPORT OF A CASE

A man about 50 years of age presented himself for inflation of the eustachian tubes, acting upon the suggestion of his physician, whom he consulted for the sudden marked loss of hearing in both ears of recent onset. Examination revealed greatly reduced hearing in both ears for the low tones with marked retraction of both ear drums. The pharynx showed evidence of a clean tonsillectomy with no lymphoid tissue in the fossae. Both posterior pillars showed large, nodular, elongated smooth masses on their dorsal surfaces.

Upon elevating the soft palate a very large mass was disclosed which almost filled the nasopharynx. Thereupon it was learned that during a recent routine general physical examination in one of the large clinics a biopsy was made from the mass on the posterior pillar, and the patient was told later that inasmuch as the specimen showed no signs of malignancy the thickening of the pillars was of no clinical importance. Now, either the mass in the nasopharynx escaped the examiner, or else it was not present at the time. In the latter case one would have to conclude that the enlargement in the nasopharynx was exceedingly rapid in its development.

It was impossible to get a catheter into the nasopharynx to inflate the tubes. A general physical examination was requested. This revealed no enlargement of the spleen or the liver, no lymphadenopathy of the nodes accessible to palpation. The chest was found normal roentgenologically, and by physical examination. The blood count, including the differential count, was normal.

The patient continued to complain a great deal about his deafness, the discomfort in his ears, and difficulty in breathing. A small biopsy from the nasopharynx revealed normal appearing lymphoid tissue. He was referred to the x-ray department, where he received two x-ray treatments. In the meantime it was decided to remove the mass from the nasopharynx surgically in order to relieve the tubal occlusion. This was done under local anesthesia with sharp currettes, a large adenotome, and sharp dissection. The masses of tissue were dissected from the posterior pillars.

Microscopic examination of the tissue revealed a great increase in the number of follicles, and a great increase in the size of individual follicles. In other words, there was a "numerical dimentional hyperplasia of the follicles," together with an interfollicular fibrosis in certain areas. Most of the follicles were separated one from the others, but appeared distorted in outline. Some were perfectly ovoid in shape. The cells of the follicles may be described as hypochromatic embryonal cells of the large lymphocytic type.

This case differs from those reported previously in that the hyperplasia appeared to be limited to the nasopharynx, and to the faucial pillars.

30 NORTH MICHIGAN AVE.

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XXXVIII

BRONCHOSCOPY AS A TREATMENT OF POSTOPERATIVE ATELECTASIS*

(Report of 90 Cases)

Joseph A. Perrone, M.D.

Pittsburgh

In 1936 the author reported 55 cases of postoperative atelectasis.[1] Since that time 35 other cases have occurred and a report of 90 cases is attempted in this paper. In scanning the literature concerning postoperative atelectasis in 1936 and again at this time, I am still amazed at the rarity with which the bronchoscopic treatment of postoperative atelectasis is mentioned. Objections of many to this treatment is that the procedure is too strenuous and risky on a recently operated patient. This fear can be completely forgotten if the procedure is done by a trained bronchoscopist. The procedure is by no means strenuous as the actual operating time is only two or three minutes.

Although atelectasis was clearly described as early as 1890, it has only been in recent years that its true frequency has been fully appreciated. Improved roentgenologic technique and the publications of obstructive atelectasis have stimulated both internists and surgeons, with the result that postoperative atelectasis is now a definite entity with definite clinical and roentgenologic evidence. As to etiology of atelectasis, this has been written in detail numerous times, so most of us are well acquainted with the various manners of its development.

There are between 4,000 to 5,000 surgical procedures performed at the Mercy Hospital, Pittsburgh, Pennsylvania, yearly. In a six-year period, 90 cases of postoperative atelectasis have occurred. Sixty-five of these patients were male and 25 were female. Cases of atelectasis were noted in all groups, the youngest being age 7 and the oldest, age 85. Nine cases occurred between ages 7 and 15, fifteen between ages 15 and 30, forty between ages 30 and 50, and twenty-six between ages 50 and 85.

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*From Bronchoscopic Clinic, Mercy Hospital, Pittsburgh.

In Table I a tabulation of the various types of operative procedures that were complicated by development of atelectasis are shown. This bears out the previously mentioned observation that atelectasis most commonly follows operations in the upper abdomen, particularly those upon the stomach and biliary tract.

TABLE I

PULMONARY ATELECTASIS FOLLOWING VARIOUS SURGICAL PROCEDURES

TYPE OF OPERATION	NUMBER OF CASES	PERCENTAGE
Hernia (inguinal)	4	4.4
Hernia (epigastric)	1	1.1
Cholecystostomy	1	1.1
Cholecystectomy	30	33.3
Gastrojejunostomy	20	22.2
Appendectomy	15	16.3
Intestinal obstruction	2	2.1
Nephrectomy	2	2.1
Cesarean section	1	1.1
Prostatectomy	3	3.3
Exploratory laparotomy	1	1.1
Tonsillectomy and Adenoidectomy	2	2.1
Perforated duodenal ulcer	2	2.1
Gastrectomy (partial)	5	5.1
Salpingo-oophorectomy	1	1.1
Total	90	

Table II shows the variable number of anesthetics used in this series of cases. Although gas and ether were the most common anesthetics, it is evident from Table II that atelectasis follows any type of anesthesia, even spinal and local. This tends to prove that no patient is free from the hazard of postoperative atelectasis regardless of the anesthetic used.

TABLE II

RELATION OF PULMONARY ATELECTASIS TO TYPE OF ANESTHETIC USED

TYPE OF ANESTHETIC	ATELECTASIS	PERCENTAGE
Ether (open drop)	25	28
Gas and ether	47	52.1
Spinal	3	3.3
Local and gas	1	1.1
Local	2	2.1
Avertin and ether	4	4.4
Avertin and gas	5	5.4
Avertin	1	1.1
Gas	2	2.1
Total	90	

The time of onset of atelectasis following surgery is shown in Table III. Seventy-four of the cases occurred between 36 and 48 hours following operation. Three cases occurred less than 12 hours postoperatively, thus showing that atelectasis can occur during the operative period. Two cases occurred three days postoperatively, and one did not occur until the sixth day. This case had all the clinical and roentgen-ray findings similar to the ones occurring 36 to 48 hours postoperatively.

TABLE III

TIME OF DEVELOPMENT OF PULMONARY ATELECTASIS FOLLOWING SURGICAL PROCEDURE

PERIOD FOLLOWING OPERATION	NUMBER OF CASES	PERCENTAGE
Less than 12 hours	3	3.3
12 hours	2	2.1
24 hours	8	8.9
36 hours	30	33.3
48 hours	44	48.9
3 days	2	2.1
4 to 6 days	1	1.1
Total	90	

The sites of election for atelectasis to occur in the various lobes of the lung are clearly demonstrated in Table IV. Thirty-five cases occurred in the right lower lobe. Massive atelectasis occurred in 17 cases. The anatomic peculiarity of the lungs and the bronchi is the reason offered for the more common occurence of this condition in the right lung.

TABLE IV

SITE OF ELECTION OF PULMONARY ATELECTASIS

SITE	NUMBER OF CASES	PERCENTAGE
Right lung (massive)	17	18.9
Left lung (massive)	8	8.9
Right lower lobe	35	38.9
Right lower and middle lobe	23	25.4
Left lower lobe	7	7.6
Total	90	

The earliest manifestation of pulmonary atelectasis is the elevation of the temperature, which in the first few hours may fluctuate between 99 and 100 degrees F. and then suddenly rise to 102 to 103 degrees F. Seven of the patients reported in this series had a

temperature of 105 degrees F.; 20 had temperatures varying between 103.5 and 104 degrees F.; the remaining ones had temperatures varying from 101 to 103 degrees F. It is amazing to note that one of the patients with a temperature of 105 degrees F. and 12 with temperatures of 103 and 104 degrees F. respectively, had only the right lower lobe involved, thus showing that the temperature is not caused by the amount of lung involved but rather by the amount of infection present. The characteristic respiratory symptoms observed in the order of their frequency were dyspnea, cyanosis, rapid pulse, and chest pain. These are caused partially by the sudden mediastinal displacement resulting from the fall in intrapleural pressure on the involved side. As the patient becomes better adapted to his intrathoracic disturbance, the respiratory embarrassment subsides. A gradually developing atelectasis disturbs the patient to a lesser degree, because there is an absence of sudden alteration in the intrapleural pressure and the vital capacity. This explains the severity of the respiratory symptoms in some cases and the relative mildness in others.

Contrary to general opinion, the early diagnosis of postoperative atelectasis is at times dependent more on the physical examination than on roentgen-ray findings. This is especially true in cases in which minor degrees of atelectasis occur and in those in which the involved area lies within the heart shadow on the left side. Expansion of the chest is limited or absent over the affected area. Often the interspaces appear retracted; the heart and trachea may be drawn toward the affected side. Often there is early hyperresonance over the affected area, but as the secretion increases dullness will be noted. At first the breath sounds are absent or diminished with no rales; later there is tubular breathing with moist bubbling rales. Positive chest findings were elicited in every patient examined after the onset of the first symptoms. On examination the heart was definitely shifted in 53 cases, questionably in 25 cases, and in 12 cases no displacement could be demonstrated.

The roentgenologic findings in the early cases of atelectasis may reveal little or no increase in density over the lung; later the shadow may be dense or homogenous. In more sèvere cases there is elevation of the diaphragm on the affected side, narrowing of the intercostal spaces, and displacement of the trachea, heart and mediastinum to the affected side. Any one of these signs may stand out predominantly over the others. If an obstructive emphysema is present, the position of the diaphragm is often lower than normal and the heart is displaced to the opposite side. Scattered bronchial

obstruction results in a patchy atelectasis, which often simulates a bronchopneumonia on the roentgen-ray film. When this type of atelectasis is encountered, there will be an absence of heart and mediastinal displacement if the sum of atelectatic area is of insufficient pressure. In 75 of these cases the roentgen-ray findings were typical; in five the diagnosis was questionable from a roentgen-ray standpoint. In the remaining 10 cases, films were not taken as the clinical picture and physical findings were typical of the condition.

It is possible to prevent postoperative atelectasis in a number of cases if a few prophylactic measures are adhered to prior to operation. Measures should be adopted to prevent drainage of secretions into and throughout the tracheobronchial tree, also obviating conditions favoring collection of secretions within the bronchi. Patients with a postnasal drip and those recovering from a rhinitis should be treated by a rhinologist. If poor oral hygiene exists, it should be treated in order to decrease the bacterial count of buccal secretions which are likely to be aspirated. The only exception to this preoperative care is in the case of emergency surgery. Patients suffering with a lung abscess, bronchiectasis, or purulent tracheobronchitis should have a bronchoscopic aspiration before operation.

During the operative procedure the use of the suction for removing secretions that have collected in the pharynx, nasopharynx and trachea cannot be overemphasized, as it diminishes secretion which would otherwise be aspirated into the tracheobronchial tree.

If an inhalation anesthesia is employed, a light narcosis should be used whenever possible in order to preserve the cough reflex. Many times coughing can dislodge mucus which has collected in the trachea and larger bronchi and thus prevent a possible atelectasis. Cyanosis under anesthesia should be reduced to a minimum. This can be prevented by the proper inhalation mixture. The use of CO_2 induced hyperventilation for de-anesthetizing on the operating table following inhalation anesthesia is a great aid, but by no means does this prevent atelectasis. This is done routinely on the surgical services of Mercy Hospital. This procedure, however, has not been used following spinal and local anesthesia. There is no definite evidence that the length of time the patient is on the operating table has any marked effect upon the incidence or extent of atelectasis. In this series of cases, atelectasis occurred with almost equal frequency among both the long and short operative procedures.

The postoperative positions of the patient may be a factor in the production of atelectasis. The tendency is to keep the patient

flat in bed with the head dependent and feet elevated. It has been demonstrated that with the head dependent the vital capacity is greatly reduced and respiratory movements are impeded. The same is true to a lesser degree when the patient is flat in bed. Therefore, it is evident that the position for least impediment of respiratory movements and for a greater vital capacity is the Fowler position. Keeping the patient in the Fowler position whenever possible and frequent changing of the position of the patient cannot be stressed too strongly.

In the postoperative treatment the effect of drugs is worthy of note. Our anxiety to give our patients a painless postoperative convalescence has resulted in the giving of narcotics and sedatives frequently and in large doses. The result has been that we have produced atelectasis in many patients, who probably otherwise would have handled the pulmonary involvement with ease, by diminishing the respiratory movements and the vital capacity and by diminishing or completely abolishing the cough reflex. The use of tight dressings and binders should be minimized if possible. Abdominal distention should be prevented. Dilatation of the stomach should be avoided by using the nasal tube at the first sign of gastric distention.

As soon as the diagnosis is made, treatment should be immediately instituted. A plan which we have found to be quite satisfactory is to stop all sedatives, encourage the patient to cough, and move him from side to side in bed. In conjunction with this, we have given CO_2 inhalations. This procedure is followed, and the patient is kept under observation for eight to ten hours. If any improvement is to take place, we have found that it usually occurs within this time. If no drop in pulse and temperature or improvement of physical signs occur, we then perform a bronchoscopic aspiration in order to remove the mucus plug. This procedure is done in bed. The only equipment needed is a bronchoscope, a laryngoscope, short and long aspirators, and a bronchoscopic aspirating pump. Prior to bronchoscopy the patient should have a tight abdominal binder if the atelectasis has followed an abdominal procedure. A small dose of morphine should be given in order to relieve the anxiety of the patient. The back rest of the bed is raised enough to have the patient in a sitting or near sitting position. The assistant stands to one side of the bed with the bite block on either his right or left thumb, depending upon which side of the bed he is standing. With the bit block on his thumb he is able to keep the patient's shoulder down with his forearm. His other hand keeps the other shoulder flat against the back rest. The operator stands at

the head of the bed, usually upon the block used as a foot rest during the regular procedure of performing a bronchoscopy in the recumbent dorsal position. The procedure in this manner at first may be a little awkward for the operator, but once done it will not be found very difficult.

The results of bronchoscopic aspiration in atelectasis has been very marked. In the majority of cases there is a sudden drop in the temperature and in the pulse and respiratory rates. This is clearly shown in Table V. The patient almost immediately feels relieved of dyspnea and is able to cough up any secretion which has not been aspirated with ease. Marked changes take place in a few hours. This has been proven by Roentgen-ray and physical examinations.

The characteristic sputum in most cases is thick, pearly, yellow, and tenacious. If placed in a container, it adheres firmly to it when inverted. The other type which is thinner in consistency and purulent may be the sputum which produces a patchy atelectasis. In all of the sputums removed from this series the bacteriologic findings showed pneumococci of some type. The largest number were type IV.

TABLE V

TIME OF DROP IN TEMPERATURE AND PULSE AND RESPIRATORY RATES FOLLOWING BRONCHOSCOPY

PERIOD FOLLOWING BRONCHOSCOPY	CASES	PERCENTAGE
12 hours	58	64.2
24 hours	22	24.8
36 hours	3	3.3
Died from other causes:		
Pneumonia	6	6.2
Peritonitis	1	1.1
Total	90	

In this series of cases all patients made uneventful recoveries following bronchoscopy with the exception of seven. One died of peritonitis, and at the necropsy the lung was found to be normal. The remaining six died of pneumonia, which is the most common complication following atelectasis. Only one case had to be bronchoscoped twice, and this would not have occurred if the patient had been a little more co-operative.

CONCLUSIONS

1. The predominating postoperative pulmonary complication is atelectasis, which may involve a portion of a lobe, a whole lobe, or whole lung.

2. Early recognition of postoperative pulmonary atelectasis is important for two reasons: (a) It allows an earlier institution of treatment and facilitates a more prompt return of the atelectatic lobe to normal; (b) it obviates the dreaded postoperative pneumonia, which may occur in atelectasis of longer duration.

3. Bronchial obstruction is the most important factor in producing atelectasis. Its removal can be achieved most satisfactorily by bronchoscopic aspiration. Once the obstruction is removed, the lung reinflates.

4. In some cases CO_2 may be of aid, but it is well to remember that just as much harm can be done. The result of CO_2 hyperventilation rests purely upon a thorough knowledge of the anatomy of the tracheobronchial tree.

5. Bronchoscopic aspiration can be done in bed. If performed by a trained bronchoscopist, there need be no fear of unforeseen complications.

6. Patients receive almost instantaneous relief followed by a marked drop in temperature and pulse and respiratory rates. Physical and roentgen-ray examinations reveal a marked change in the involved area in a short time.

7. Ninety cases are included in this series.

MERCY HOSPITAL.

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Clinical Notes

XXXIX

EPIDERMOID CARCINOMA OF THE NASOPHARYNX OCCURRING IN TWO YOUNG BROTHERS

WM. D. STINSON, M.D.

MEMPHIS

Cancer of the nasopharynx is not rare, and excellent articles on the subject appear frequently in literature. The coincidence of its occurring in two brothers within the span of two years is, however, unusual and tragically dramatic. It was unquestionably the most depressing incident in my professional experience. What consolation can there be for a 17-year-old boy who attends his young brother through a year of slow death, and then suddenly realizes that he himself is caught within the jaws of the same fate?

There is no necessity for detailed consideration of carcinoma of the nasopharynx in this case report, though some generalization is in order for those of us who are somewhat vague in our knowledge of pathology.

Pathologists generally give three main histological varieties of epidermoid carcinoma, the squamous type, the transitional-cell carcinoma, and basal-cell type.

These cases being of the transitional-cell type, I shall quote directly Ewing's[1] description of them in his book "Neoplastic Diseases":

> "Certain mucous surfaces are covered with stratified epithelium, of which the cells are delicate, do not possess spines, do not produce keratin, and show little capacity to hornify. This type of epithelium is found over the tonsils, the vault of pharynx, nasal passages, base of tongue, and the nasal sinuses. It often covers deposits of lymphoid tissue, and hence has sometimes been called lympho-epithelium."
>
> "Tumors of this type of epithelium present histological and clinical peculiarities, which seem to call for their recognition as a special subvariety of epidermoid carcinoma. They are more closely related to squamous than to basal-cell carcinoma, and it is possible that very anaplastic and malignant tumors derived from adult squamous cells may exhibit the same clinical features."

"The transitional-cell tumors arise in the locations mentioned, but do not, as a rule, produce bulky growths. Their tendency is to yield small infiltrating growths which are easily overlooked, but which early invade the lymph-nodes draining the affected area, so that the first sign of the disease is generally an enlargement of the cervical nodes. Very careful search for the original tumor has often been unsuccessful for many months, so that the diagnosis of primary tumors of the nodes is often made, or branchiogenic carcinoma is often suspected. Branchiogenic carcinoma, however, is almost always squamous. The disease extends slowly from one node to another, and in many cases the extensions pass the regional nodes, and reach the liver, lungs, bones, and mediastinum. Many cases occur in young subjects in whom the suspicion of carcinoma is not readily accepted. The course of the disease is often prolonged."

"The structure shows sheets of large, pale, delicate cells with thin membranes, large nuclei, and indefinite cell outline. They infiltrate the submucosa or lymphoid tissue in sheets or small groups, or singly. The diagnosis of primary endothelioma is often suggested. In the nodes the cells may reveal some traces of squamous characters, or they may become less differentiated and the structure may resemble lymphosarcoma. In the hepatic metastases all traces of epithelial character may be lost, and the tumor grows as a diffuse, round-cell process. Abundant admixture with lymphocytes obscures the histological picture, and complicates the diagnosis in many primary and secondary tumors."

"Transitional-cell carcinoma of the nasopharynx has been recognized by many authors, but has never been sufficiently emphasized. Crowe and Baylor, and Jovin report several characteristic cases with excellent illustrations. New observed several cases, and noted the difficulty of detecting the original tumor. Quick and Cutler collected a series of cases at the Memorial Hospital, and described in detail the structural and clinical peculiarities. It should be noted that surgical results in this type of epidermoid carcinoma are poor owing to numerous and widespread metastases, while the undifferentiated character of the cells renders them highly susceptible to radiation. The primary tumor and the cervical metastases may often be cleared up by heavy external radiation, but most of the cases eventually succumb to internal and distant metastases."

Boyd[2] differs in dividing transitional-cell carcinomas and lympho-epitheliomas into specific types, calling especial attention to their similiarity to lympho-sarcoma and concluding:

"It may be pointed out that much of what has been said under this heading may be summed up by saying that in the mouth and pharynx there occurs a group of highly anaplastic radiosensitive epidermoid carcinomas characterized by the inconspicuous character of the primary lesion, the massive involvement of regional and distant lymph nodes, and the formation of visceral metastases. The more anaplastic the carcinoma, the greater becomes the difficulty of distinguishing it from lymphosarcoma."

CASE 1.—J. W. Baptist Memorial Hospital. Age 10, white, male. Admitted to the hospital July 3, 1937. Five months previously he began complaining of a sore throat which persisted for four months, at which time a swelling appeared in the left side of the neck. There was occasional fever and expectoration of blood,

with intermittent headaches. Tuberculin test done at that time was negative. A tonsillectomy was performed without improvement.

Examination on day of admittance by the interne was negative except as follows: "All the glands on the left side of the neck are greatly enlarged, firm, only slightly tender. No redness or increased heat. The glands are movable." The eye reflexes and pupils were normal. The throat was normal but no notation was made of the nasopharynx.

A biopsy on the glands of the neck was done by Dr. E. G. Kelly on July 8, 1937. The pathological report was: "Undifferentiated metastatic carcinoma." X-ray of the skull, cervical spine and lungs failed to reveal any metastases.

July 20th, an examination of the nasopharynx by Dr. J. D. Evans under general anesthetic revealed a raised indurated mass in the left nasopharynx which bled freely. A specimen was taken, the report on which was: "Transitional cell epithelioma."

He was given nineteen consecutive daily treatments of 200 R x-ray therapy, the exposure being varied over the right and left sides of the face to include the nasopharynx and neck. He returned in December unimproved and his local physician reported that he finally died May 17, 1938.

It is a sad commentary that this child was subjected to a tonsillectomy and five months of observation without an adequate examination of the nasopharynx.

CASE 2.—L. E. Baptist Memorial Hospital. Age 17, white, male. Brother of patient Case 1. March 29, 1939, this youngster came to the receiving ward of the Baptist Memorial Hospital and asked pitifully if he had the same thing his brother had. He gave a history of a sore throat several weeks previously, followed by a swelling in the right side of the neck.

There was a large firm nodule in both the anterior and posterior cervical lymphoid chain on the right side. Examination of the nasopharynx through the nose revealed a large mass of adenoid tissue. Springing from the anterior surface of the adenoids was a papillomatous, almost pedunculated, firm tumor about a centimeter in diameter, which bled freely.

X-ray of the chest was negative. Lateral view of the pharynx showed an irregular flat projection about two and one-half centimeters in length in the posterior wall of the nasopharynx. Under

general anesthesia, the entire nasopharynx was cleaned out. The biopsy report on the tissue submitted was as follows: "The section in the superficial portion shows sharply limited surface epithelium and a diffuse growth of lymphoid cells. There is one prominent follicle and a cyst-like space containing eosin staining material. The deeper portion of the section is invaded by strands and masses of small, pale staining, granular cells which are apparently transitional in type—a few mitotic figures noted. Diagnosis: Transitional-cell carcinoma."

He was given sixteen consecutive daily treatments of 200 R x-ray with varied exposure over two sides of the face and neck.

June 26, 1939, the nasopharynx was clean, the membrane thin, and showed no evidence of any growth. The posterior gland had disappeared but there was a small anterior cervical gland, barely palpable. Four exposures of 200 R each were again given.

January, 1940, he wrote that he had had no further trouble with his nose, but was confined to his bed with severe pain in his leg which was draining pus—probably a metatasis.

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XL

REPORT OF TWO CASES OF PEDUNCULATED OSTEOMA OF THE EXTERNAL AUDITORY CANAL*

W. G. KENNON, M.D.

NASHVILLE

Osteomata of the external auditory meatus are mentioned so casually and briefly in the textbooks which I have consulted, that it would appear that they are of everyday occurrence.

In reviewing the literature on this subject from 1925 through 1938, I was able to find only five references; three of them from American sources. Those from the American sources were by Lillie and Williams,[1] Lewis[2] and Vail.[3] In Lillie and Williams' case, which was very similar to the two I am reporting, the diagnosis was verified by microscopic examination. Lewis' case which, from the description, was also similar was treated by dietary measures and the growth disappeared. In this case, however, the character of the growth was not determined by biopsy. Reports of somewhat similar cases were found in the foreign literature by Banyai and Janota[4] and by Aprile. [5]

Vail's paper is by far the most complete and informative and is difficult to abstract as one is tempted to copy it verbatim. He quotes from various textbooks on otology and pathology and from papers on archeology. It appears that osteomata have been found to occur very frequently in prehistoric skulls from excavations in Peru and in North America. Males are more frequently affected than females. Vail suggested that the term "exostosis" be reserved for new bone formation which results from some form of irritation, and the term "osteoma" be restricted to new bony growths which develop without any form of irritation.

CASE 1.—Mrs. W. C. T., aged 21, white, was first seen on June 9, 1938. She complained that she had noticed a growth in the left external auditory canal for about a year. At times there were

*Read before the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Columbia, South Carolina, January 8, 1940.

slight deafness and a feeling of stuffiness in this ear but no pain. Her previous medical history has no bearing on the case. The physical examination was negative except for the local condition. Blood Wassermann test was negative. Examination showed a small, hard growth covered with skin apparently attached posterosuperiorly by a small pedicle. This growth seemed to be slightly movable, but I could not be sure of this on account of the patient's sensitiveness.

On July 13, 1938, under general anesthesia, the patient having been prepared as for a mastoidectomy, a small curette was passed anteriorly between the canal wall and the tumor, which was lifted out. The canal was tightly packed with a gauze wick, which was removed the following day. The skin wound healed within a week and examination at this time showed the canal, drum membrane and hearing to be normal.

Pathologist's Report: Histological sections from decalcified tumor show on one border a small portion of epithelium, beneath which there is a fibrous layer which appears to be periosteum. Beneath this fibrous layer there are bony trabeculae, growing in an orderly fashion, and congested Haversian canals. There are areas showing remnants of calcified cartilage, but in no area is there evidence of a malignant process.

Diagnosis: Osteoma.

CASE 2.—Mr. T. D., aged 25, white, first seen June 9, 1939. He had noticed a growth in the left external auditory canal for six months. For the past month there had been some deafness in this ear. There was no pain. His previous history had no bearing on the complaint. His physical examination, other than the ear condition, was negative. The blood Wassermann test was negative. The right ear was normal. The left ear showed a growth almost occluding the lumen of the external auditory meatus. This growth was covered with normal skin and was not unduly sensitive to palpation with a probe. It appeared to be definitely pedunculated with the attachment to the posterior portion of the canal wall. It did not seem to be movable.

On June 15, 1939, the growth was removed in the same way as in Case 1. The only difference between the two growths on gross examination was that in the second case there was a very small bony attachment, the bony pedicle having a diameter of about 2 mm. This patient's postoperative course and convalescence were as uneventful and the canal and hearing as normal as in Case 1.

Pathologist's Report: The specimen is an ivory-hard spherical nodule about 0.6 cm. in diameter. Its surfaces are smooth except at one point where there is a short pedicle about 0.2 cm. in diameter.

The nodule is covered by thin atrophic squamous epithelium which is separated from a mass of compact bone by a thick layer of fibrous connective tissue. Within the compact bone there are numerous, roughly spherical, marrow spaces which contain relatively few marrow cells. Most of them contain a few osteoblasts which are closely applied to the bone. No cartilage is observed.

Diagnosis: Compact osteoma.

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XLI

THROMBOPENIC PURPURA AND THE ROLE OF X-RAY TREATMENT TO THE SPLEEN

EDWIN D. WARREN, M.D.

TACOMA

Because of the frequency with which the rhinologists see persistent epistaxis, I believe they should have a better understanding of purpura and its modern conception.

As a text for the discussion of the subject of thrombocytopenic purpura, I should like to present the following case report:

A. G., an adult male, aged 49, had the first warning of any abnormality of his blood clotting on April 25, 1938. While playing bridge he had a profuse bilateral nasal hemorrhage. After attempting for a few minutes to control it with cotton packing in the anterior nares his friends called his family physician who attempted to pack it anteriorly. The blood ran down his throat and choked him. He fainted while on the way to the hospital and twice while in the hospital from rapid exsanguination. Upon reaching the hospital he was given 20 cc. coagulen ciba intramuscularly and the nasal hemorrhage was controlled this time rather easily with stryphonon gauze which is a Vienna preparation of gauze impregnated with cephalin from ox brain tissue. Up to this time it was thought he might have an ordinary epistaxis as he had never experienced a similar catastrophe; however, it was noted that the hemorrhage was occurring from several areas at the same time. As soon as the blood work could be studied it was found his clotting time was 20 minutes and the bleeding time 10 minutes. The platelet count was 113,500. There were no other noteworthy changes in the blood except secondary anemia. That night he was given a Peck test which consists of an injection of 0.1 cc. of 1:3000 moccasin venom. Since this was negative as indicated by lack of an ecchymotic area or hemolysis at the site of injection, he was given 0.4 cc. of moccasin venom. The patient bled very slightly at intervals for three days. The hemor-

rhage was always easily controlled with the stryphonon gauze. It was not necessary to resort to a posterior pack.

On April 29, 1938, after one dose of snake venom, the clotting time was four minutes and the bleeding time was two and a half minutes. The platelet count was 135,000. The spleen and the liver were not enlarged. The blood pressure was 144/88. The physical examination was entirely negative.

The family history was carefully gone into and was entirely negative as far as blood dyscrasias were concerned. The patient was cognizant of his family history for two past generations.

This man had an uneventful life, except that he worked extremely hard as a dentist, and at great nervous tension, up until January, 1940. He always took two ounces of whiskey on arrival at home, which he said gave him relaxation and made him enjoy his dinner. On week-ends he consumed more but was by no means a heavy drinker. This is mentioned because of the possibility of the effect of toxins on the spleen.

He was given snake venom two or three times weekly for six weeks, working up to and maintaining a dose of 1 cc. except when he showed the reaction of sensitization. Then it was dropped back and he was desensitized by small ascending doses.

On January 13, 1940, while playing bridge on a week-end after several drinks, he had an extremely severe hemorrhage from both sides of the nose. He was taken to Tacoma General Hospital immediately and given 20 cc. of coagulen and both nares were packed. He bled profusely through the packing on both sides. Finally posterior packs were inserted on both sides which controlled the situation. The next morning a blood count revealed 110,000 platelets, a clotting time of twenty-six minutes and a bleeding time of eight minutes. Due to the pressure of the packing the patient developed an acute purulent bilateral sinus infection and the right ear-drum ruptured spontaneously. It discharged for ten days when it spontaneously ceased. Because of this obstacle the packing was removed but the patient bled again within an hour after the packing was removed. It was reinserted but bleeding persisted. He was given several ampules of coagulen, 1 cc. of moccasin venom and repeated doses of koagmin. His clotting time was reduced to eighteen

minutes. The clotting time, bleeding time and platelet count were taken at intervals.

Because of the extremely poor condition of the patient it was considered inadvisable to remove the spleen, as his hemoglobin was reduced to 39 per cent on January 24, 1940. Successful references to treatment by x-ray were found in the literature. He was given 200 r units over the spleen on January 24th, and the same dose repeated on alternate days until he had a total of 100 r units. An immediate rise in platelets from 108,000 to 210,000, was obtained and on the following day it was 270,000. There was no more bleeding after second dose of x-ray.

As soon as possible a blood transfusion of citrated blood was given. After this the patient bled more persistently than ever. A massive dose (4 cc.) of snake venom was given without unfavorable reaction. The patient ceased to bleed and left the hospital on January 16, 1940. The next day a prothrombin time test was done and was found to be decreased 30 per cent. This seemed a definite indication for vitamin K, so this was given along with Bilen. The next day he bled profusely again at midnight. He was packed in the home and the bleeding stopped readily. The packing was removed one hour later because of the bad sinus infection, and he bled again at 3:00 a. m. He bled all that night and the next day (January 20, 1940) until noon in spite of the tight packing and two blood transfusions. Such episodes persisted for the next week until the hemoglobin was down to 32 per cent. In the meantime he was given six blood transfusions. The clotting time was finally reduced to seven minutes and the platelets came up to 225,000, and the patient was able to leave the hospital on January 31, 1940.

A greater response of platelets and control hemorrhage was obtained after the first dose of x-ray to the spleen than from any other treatment. Of the various things given to help assist in getting the blood to clot was coagulen ciba, a total of 140 cc. in eleven days; koagmin, ten doses of from 0.5 cc. to 3 cc. (some given intravenously and some subcutaneously), vitamin K with Bilen (three of each three times daily), local and intramuscular fibrinogen, and local and intramuscular thromboplastin; large doses of cevitamic acid (vitamin C); large doses of calcium and a liver and jello diet. He was given six blood transfusions and four intravenous injections of saline and glucose when his blood pressure was too low.

At no times were there hemorrhages from other places and he did not have petechia hemorrhages or blue spots on the extremities.

The clot reaction was slightly prolonged, three hours on January 15, and four hours on January 16. The fragility test was as follows:

January 16, 1940	—	Patient begins	0.36
		complete	0.30
		Control begins	0.38
		complete	0.30
January 21, 1940	—	Patient begins	0.44
		complete	0.38
		Control begins	0.42
		complete	0.32

The tourniquet test was negative. The temperature reached 102 degrees at the maximum which was explained by the purulent sinusitis and the otitis media.

He has had an uneventful time since leaving the hospital on January 31, 1940. The platelets, hemoglobin and red cells are steadily increasing. He is being given x-ray treatment to the spleen, which will be stopped when he has a total of 800 r untis, and he is still receiving tri-weekly injections of moccasin venom. A sternal puncture was performed by Dr. Larsen on January 30, 1940.

COMMENT

This case is particularly interesting, as it illustrates the necessity of close observation to prevent such episodes. It demonstrates the value of x-ray therapy. It shows the possibility of a blood dyscrasia without any pathological familial background. It brings up the question of toxic agents such as alcohol on the spleen and the bone marrow. This case was one of an abnormal clotting mechanism but of no increased capillary permeability or fragility. In the type where capillary permeability and fragility are present, large doses of calcium and vitamins help. In this type of case they are useless. Purpura has been classified in many different ways. More recent and enlightening studies which consider the pathology of the bone marrow, lead to a better classification.

Symptomatic Purpura: This is primarily due to capillary disease. The blood platelets are normal. The megakaryocytes are increased in number in the bone marrow (160 to 180 per 18 mm. square), but are less than in thrombocytopenia purpura. The bleeding time, clotting time and clot retraction are normal. The tourniquet test is positive. The anemia is usually normocytic in type.

The condition is often based on a toxic origin, so there are often seen toxic granulocytes.

Essential Thrombocytopenia Purpura (active phase): There is bleeding from the gums, nose, bowel or skin. Petechiae are present. The blood platelets are absent or greatly decreased. There is an increase in megakaryocytes in the bone marrow from 900 to 1,000 per 18 mm. square. Following splenectomy there is cessation of bleeding. When an acetone extract of the spleen is made and injected into a rabbit there is a rapid reduction in the platelets and hemorrhages occur, as demonstrated by Troland and Lee.[1]

Chronic Thrombopenia Purpura (secondary type): In this the platelets are reduced, the bleeding time varies from five to ten minutes. The clotting time varies from six to thirty minutes. The clot retraction may or may not be reduced. The fragility test is usually but not always positive. Removal of the spleen does not necessarily stop the bleeding episodes as shown by Limarzi and Schleicher.[2] It is often precipitated by infections or toxins. The megakaryocytes in the bone marrow are from 20 to 30 per 18 square mm. This type probably should be treated with moccasin venom and x-ray therapy until the patient is in proper condition for a splenectomy. It should never be done when the patient is a poor surgical risk.

Conditions Resembling Purpura: There are many conditions such as leukemia, Hodgkins disease, agranulocytosis, infectious mononucleosis, polycythemia vera, and aplastic anemia in which bleeding may occur and be difficult to stop and which at first may appear to be purpura. In chronic recurrent epistaxis one should have a complete blood count, bleeding time, clotting time, clot retraction test, platelet count, fragility test, prothrombin time and bone marrow study to make a proper classification of the case. Of the above mentioned the bone marrow study offers the most information. When there has been a loss of blood in the above mentioned conditions the blood picture is often so altered as to be confusing. The platelets are exhausted; there is an effort on the part of the bone marrow to regenerate the blood elements and many immature cells make their appearance.

Hodgkin's disease is the only one of the above in which there is an abnormal number of megakaryocytes in the bone marrow anywhere approaching that in thrombopenic purpura, except very rarely there may be a decided increase in megakaryocytes in the bone marrow in polycythemia vera.

Bone marrow studies definitely help in suspected aplastic anemia and leukemia. In the former condition there is a depression or a loss of megakaryocytes in the bone marrow, while in the leukemias erythrogenesis is absent. Erythrogenesis is most marked in the hemolytic anemias, Neumann-Pick's disease, sickle-cell anemia and Cooley's anemia.

Bone-marrow studies are usually made by sternal trephines or punctures with a large blunt needle according to the method described by E. Osgood in his Atlas of Hemotology.

MOCCASIN VENOM

This was first introduced by Peck and Rosenthal[3] in 1935. A 1:3000 dilution of moccasin venom is used. The patient should be tested for sensitivity and if not sensitive, 0.4 cc. should be given and rapidly increased to a maximum dose of 1 cc. The action is apparently on the blood vessels more than on the platelets; however, it does assist in increasing platelets slightly. It is more beneficial for many other hemorrhagic conditions than for thrombopenic purpura. Since its introduction some workers have kept under control 17 out of 19 patients with thrombopenic purpura over a period of years. A dose as large as 5 cc. was used by C. S. Imerman[4] in 1938 without reaction. I used 4 cc. in this case without any reaction. In fact it seemed to be the only thing that really changed the picture from what threatened to be a fatal result.

I do not believe one should depend entirely on snake venom to tide over the thrombopenic purpura for a period of years as demonstrated by this case. The spleen is the thrombocytic agent and as long as destruction is going on something must be done in regard to it.

REMOVAL OF THE SPLEEN

There is an opinion among the majority of doctors with whom I have discussed this, that thrombocytopenic purpura indicates removal of the spleen and that is all there is to it. This is a misconception. Brown and Elliot[5] reported 21 cases. Ten were treated by splenectomy. Of these ten, excellent remissions were secured in eight, one improved and one died. Of the eleven untreated patients five cases improved, five remained unimproved and one died.

In general the published results of splenectomy show an operative mortality of about 14 per cent with about 65 per cent cure.

Splenectomy may be a life saving measure when augmented by small, whole-blood transfusions. In this case, citrated blood transfusions increased the hemorrhage.

X-RAY TREATMENT OF THE SPLEEN

Mettier and Stone[6] treated six cases of thrombocytopenic purpura with fairly large doses of x-ray to the spleen and obtained remissions lasting for four years. Six to twelve doses of 200 r daily or on alternate days were given. This always resulted in good platelet response. Rudisill[7] treated eight cases of thrombocytopenic purpura with a similiar technique. Of this group seven cases showed complete remissions lasting from one to four years, one which was fatal, was complicated by acute myelogenous leukemia. He sees no justification for splenectomy when x-ray is so completely successful. Certainly in this case it was effective and it produced a rapid rise in platelets and cessation of hemorrhage. I consider it worthy of a trial but believe if the platelet count and blood picture do not remain near normal splenectomy should be resorted to.

VITAMIN K

In this case vitamin K did no appreciable good. Before using vitamin K a prothrombin time was done and found to be 70 per cent normal, so it was given along with bile salts as recommended. It is a very expensive medication and I should not care to depend on it entirely. However, I believe it should be used in the chronic cases.

It is not of much value in acute hemorrhage of this severity. Its indication is mainly as a preliminary to bile tract surgery or when there is a prolonged clotting time due to hemolytic or obstructive jaundice.

CONCLUSIONS

1. The absence of hereditary factors and the age to which he has lived through other episodes exclude the possibility of hemophilia in this case. The ready regeneration of blood excludes the possibility of aplastic anemia.

2. This case is of special interest because a comparison of response of platelets was possible during two hemorrhages. The response to x-ray therapy to the spleen was decidedly noticeable when it was used.

3. Citrated blood transfusions seem to make hemorrhage of this type worse. In shock when blood volume is low whole blood, acacia or glucose should be used.

4. The action of snake venom is on the vessel walls. The action of vitamin K is to increase the prothrombin. It is definitely indicated if the prothrombin time is abnormal.

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XLII

PRIMARY CARCINOMA OF THE EUSTACHIAN TUBE

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SAN DIEGO, CALIFORNIA

Carcinoma arising in or about the eustachian tube, without involving the mucous membrane of the nasopharynx, is an uncommon condition, which presents difficult diagnostic and therapeutic problems, and apparently bears a very bad prognosis. Brief examination of the recent literature disclosed about six pertinent articles in the foreign literature, particularly in Italian, and none in English or American literature.

During the past four years, two cases of carcinoma of the eustachian tube have been studied on the otology service of Furstenberg, at the University of Michigan. In one case, the site of the primary lesion was suspected, but not proved until autopsy. In the second case, the malignancy was totally unsuspected clinically and demonstrated only on postmortem examination.

The first patient was a 33-year-old Russian Jew (H. S.), who was first seen on May 6th, 1936. In September, 1935, he first noticed deafness and tinnitus in the right ear, associated with right occipital headache. Swelling in the right side of the neck just below the mastoid tip appeared in December, 1935.

On examination the right canal was normal, and the membrane was dull, not abnormally full, and without landmarks. There was a slight conductive deafness. The left ear appeared normal. The nasal septum was fairly straight, with an adequate breathing space. The throat and larynx were normal. It was never possible to do a satisfactory mirror examination of the nasopharynx because of poor cooperation on the part of the patient. Palpation raised the question of a small flat nodule in the right fossa of Rosenmüller. There was a large, non-tender, homogeneous swelling of the right side of the neck covering the sternomastoid muscle and extending into the posterior triangle. Two biopsies of the suspected area in the nasopharynx showed only lymphoid tissue without carcinoma. A biopsy of the mass in the neck showed a poorly differentiated squamous cell carcinoma. Roentgen studies revealed clouding of the right mas-

toid cells, some increase in the density of the soft tissue lateral to the right wall of the nasopharynx, and suggestive loss of bony structure around the right foramen lacerum. The general physical and the usual laboratory examinations contributed nothing.

The patient received 5800 r over a right cervical port in fractionated doses extending over a month's time. This produced a marked decrease in size of the cervical adenopathy. He was discharged on June 29th, 1936.

On July 23rd, 1936, he developed severe intermittent pain in the low back and epigastrium; not influenced by activity, change of position, or food. He re-entered the hospital a week later, and that day developed fever of 103 degrees. Septic temperature, night sweats and occasional chills continued. Roentgen studies of the gastrointestinal tract, gall bladder, pelvis and spine were negative. On August 10th and 11th, 600 r were given empirically to each of two ports over the lumbosacral spine with some relief of pain. Over the next three months the course was a constant declivity with almost daily elevation of temperature to 103 degrees, no leucocytosis, and no physical findings of further metastasis, aside from a few discrete nodes in the left posterior cervical triangle. The cause of the long-lasting septic temperature was not found. There was no neurological evidence of extension of neoplasm to the brain. The chest was negative on physical and roentgen examination. The urinary tract was normal. Agglutination reactions were negative. He died in coma on November 18th, 1936.

At postmortem examination a nodular mass about three centimeters in diameter was found in relation with the mid-portion of the right eustachian tube. There was no involvement of the mucous membrane of the nasopharynx. Microscopic study revealed a poorly differentiated medullary carcinoma of apparent squamous cell derivation. It involved the eustachian tube and infiltrated far about it. The ribs, sternum, vertebrae, cranium, spleen and liver all showed metastatic carcinoma.

The second patient was a 57-year-old white male (W. M.), who was first seen on April 5th, 1937. In December, 1935, he developed intermittent and later constant pain in the left ear. There had been no previous ear disease and no otorrhea accompanied the pain. No cause for the pain could be found. Early in November, 1936, a left myringotomy was done, following which there was a scanty discharge of serum and blood for less than 24 hours. The pain then increased in severity and remained localized to the left

ear. About Thanksgiving Day, 1936, a left complete mastoidectomy was done. The pain was not influenced by the surgery, and from then on the patient had a constant otorrhea. About Christmas, 1936, the mastoidectomy was revised, without influencing the pain which continued to be constant, severe, and localized to the left ear. Early in March, 1937, severe pain in and behind the left eye developed. Diplopia was first noticed by the patient on March 20th, 1937. There had been no nausea or vomiting, and no chills. Narcotic in increasing doses had been used for six months to control the pain.

When seen on April 5th, 1937, the patient appeared older than the given age of 57, and gave the impression of being chronically ill and in great pain. He complained of severe pain in the left ear, and pain which radiated over the left side of the head, particularly into and behind the left eye. Temperature, pulse and respirations were normal. The general physical examination was not remarkable. Otological examination revealed a normal right tympanic membrane and canal. The left canal contained a small amount of very foul pus. The tympanic membrane was thick and red, with a posterior perforation through which a polyp projected. There was a well healed left postauricular incision. A profound deafness was present on the left side. The nasal septum was deflected far to the left and there was polypoid change in both middle meatuses. No pus was seen. The nasopharynx and larynx were negative. The action of the facial nerve was normal, but there was complete paralysis of the left abducens nerve. Ophthalmological examination was negative aside from the abducens paralysis. Neurological examination showed hypesthesia over the sensory distribution of the left trigeminal nerve. The consultant diagnosed the Gradenigo syndrome and probable petrositis on the left with a questionable epidural abscess.

Roentgen studies of the mastoids revealed the expected operative defect on the left with some remaining clouded cells over the sigmoid sinus. There was marked sclerosis of the left petrous pyramid with some irregularity of the tip, in contrast with a pneumatized, well developed pyramid on the right. There was a hyperplastic pansinusitis which was more severe on the left, on sinus films. Blood studies at the time of admission revealed a hemoglobin of 72 per cent, Sahli; a white blood cell count of 5,050; a differential count of 65 per cent polymorphonuclears, 7 per cent eosinophils, 22 per cent lymphocytes, and 6 per cent monocytes. The Kahn test was negative.

Five days after admission a left radical mastoidectomy was done without finding any fistula leading to the petrosa. Three days later the temperature, pulse, and respirations rose suddenly to 105.6 degrees, 130, and 35. The only warning had been mild stupor for a few hours. The neurological consultant demonstrated a slightly stiff neck. There was no Kernig or Brudzinski reflex, and no paralysis. The wound was explored the same day. The sigmoid sinus felt slightly firm but still blood containing. The dura over the posterior fossa was discolored but not tense and the neurosurgical consultant did not see indication to needle the brain in search of an abscess. A lumbar puncture revealed cloudy fluid from which the hemolytic streptococcus was isolated on culture. Respirations ceased the following day, April 14th, 1937, with the patient in deep coma, temperature, pulse and respirations of 105 degrees, 130, and 25, and a white cell count of 29,850.

At autopsy it was found that a medullary squamous cell carcinoma had completely replaced the mucosa of the outer third of the left eustachian tube and had extended widely to infiltrate the petrosa, infratemporal fossa, and to invade the gasserian ganglion. There was a chronic pachymeningitis overlying the petrosa, and a widespread acute leptomeningitis. Recent thrombosis of the sigmoid sinus on the involved side was demonstrated. No metastasis to the cervical lymph nodes or to the viscera was found.

It is thought to be worthwhile to report these two completely studied cases because they are unusual, and were baffling problems in diagnosis and treatment. In the first case it was felt that the eustachian tube might be the site of the primary lesion for the metastasis in the posterior cervical nodes, since the nasopharynx was normal and examination of the ear gave evidence of obstruction of the tube. In the course of this case the long continued, unexplained fever was a prominent feature, and was finally thought to have been due to the replacement of normal tissue by neoplasm which was exceedingly widespread at postmortem examination. In the second case, the possibility of neoplasm was not considered by the otology staff and not suggested by the consultants. And yet the history alone of ear pain unaccompanied by ear findings for a year before surgical intervention and its continuation afterward, should possibly have suggested some condition other than a simple inflammatory disease of the ear and mastoid. It is doubtful that early diagnosis could have influenced the outcome in either case for neither neoplasm was surgically accessible, though intensive irradiation properly directed might have influenced the course.

It is suggested that neoplasms about the eustachian tube should be considered when there are persistent symptoms of eustachian tube obstruction in the absence of a lesion in the nasopharynx, when a primary lesion to account for metastatic cervical nodes cannot be found, and when there is ear and head pain which cannot be explained.

607 MEDICO-DENTAL BUILDING.

XLIII

POLYPOID GRANULOMA OF THE LARYNX FOLLOWING ENDO-TRACHEAI ANESTHESIA

WILLIAM A. SMILEY, M.D.

CHICAGO

In a review of the literature, I have been able to find but three cases of polypoid laryngeal disease following endo-tracheal anesthesia.

In 1932, Clausen[1] reported a patient operated on in September, 1931, under endo-tracheal, nitrous oxide-oxygen anesthesia. A soft catheter through the nose was introduced by direct intubation. In February, 1932, Negus found a granuloma at the posterior end of the larynx below the right vocal cord. A few days later the patient coughed up a small pea-sized tumor, which was composed of blood clot, with very little early organization. The larynx was subsequently examined and found normal.

Gould[2] in 1935 reported a case of subglottic granuloma following endo-tracheal anesthesia administered for three hours and ten minutes. A Magill tube was introduced blindly at the first attempt. Hoarseness developed within six weeks. Two attacks of dyspnea and cyanosis occurred about three months after anesthesia, and a subglottic granuloma was removed. Two months later a recurrence was removed. The histological picture was one of granulation tissue undergoing degeneration. There was no indication of tuberculous or neoplastic process.

Cohen[3] in 1938 reported a case of nitrous oxide-oxygen ether anesthesia of fifty-five minutes duration. A Magill tube was introduced blindly. Two months later she had a sensation of something moving in the throat. A laryngeal tumor was seen at the base of the right vocal cord. The tumor was removed a week later and did not recur. The tissue was a polypoid granuloma of unspecified nature from a vocal cord, composed of fibrous tissue with numerous capillaries lined with large endothelial cells. It was infiltrated with lymphocytes and plasma cells, and superficially, polynuclear neutrophiles covered with fibrin. No throat symptoms of any kind had

preceded the operation, so it was felt that the granuloma had been caused by the anesthetic procedure.

This patient, a well nourished, icteric and slightly cyanotic female of 35 years, had been operated on at the Michael Reese Hospital February 22, 1939, for stenosis of the common bile duct. The anesthetic used was endo-tracheal cyclo-propane. A soft tracheal catheter was introduced under direct vision. Duration of anesthetic was one hour and thirty-five minutes. Convalescence was normal except for the expectoration of slightly blood-tinged mucus during the first few hours. On March 2, the tenth postoperative day, she first complained of hoarseness. Laryngeal examination March 7th revealed a small polyp in the left side of the larynx. This patient was examined January 4, 1939, and no notation made of laryngeal pathology. By March 25, the polyp had increased in size and seemed to hang by a pedicle. Removal was advised, but the patient refused, and on March 31 was discharged to a convalescent home. On May 3, 1939, she was admitted to the bronchoscopic service of the Michael Reese Hospital. Her complaints were difficulty in breathing and inability to speak above a whisper.

Indirect laryngoscopy revealed a large polypoid swelling below the left vocal cord. It was seen to rise between the cords when the patient attempted to phonate. Lateral and antero-posterior x-rays were taken. In the lateral film a rounded shadow about one cm. in diameter was seen below the level of the vocal cords. In the anterior view with the intra-esophageal film, taken during phonation, the shadow is seen above the level of the vocal cords.

On May 5, under local anesthesia, the tumor was removed through a Jackson anterior commissure laryngoscope. It was a soft rounded mass about 1.5 cm. from above downward, and about one cm. in transverse diameter, and was pedunculated. Irregular remnants were removed, and the tissue was sent to the laboratory.

The report of the pathologist is as follows: "The tissue is lined in part by a layer of stratified squamous epithelium, and in part by stratified columnar cells amongst which are numerous goblet cells. Beneath the lining epithelium is a fairly well demarcated, densely cellular nodule which is composed of many thin-walled blood vessels lined by swollen endothelial cells and filled with red blood cells and, intermingled with these blood vessels, many elongated spindle-shaped cells with pale vesicular nuclei and pale eosinophilic cytoplasm. Throughout the tissue there is a diffuse infiltration of polymorphonuclear leucocytes interspersed with which are occasional eosinophilic

leucocytes and small and large mononuclear leucocytes. Around the periphery of this nodule there is a zone of extravasated red blood cells and adjacent to this a layer of amorphous eosinophilic material embedded in which are many polymorphonuclear leucocytes in varying states of disintegration. Microscopic diagnosis: Granulation tissue with acute and chronic inflammatory changes, (Pyogenic Granuloma)."

Recovery was uneventful, and her voice returned to normal, but on July 6, another small polypoid mass was removed. Since that time there has been no recurrence.

It seems justified to conclude that this subglottic granuloma was a result of trauma from the endo-tracheal anesthesia. Because of the microscopic pathology of the tissue removed it is suggested that the larynx may react to trauma by the formation of an infectious granuloma such as we infrequently see on the nasal septum.

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XLIV

TWO UNUSUAL CASES OF POSTANGINAL SEPSIS

E. I. Matis, M.D.

Kaunas

The question of post-tonsillitis complications is one of the most important in modern medicine, and much has been written on the subject. Among these complications, acute postanginal sepsis is the most dangerous, causing a death rate which is very high and, judging by the latest investigations, it is very prevalent in many countries. According to the figures supplied by Haymann,[1, 2] it must be agreed that postanginal sepsis is responsible for a death rate of 5.3 per 1,000 (equal, according to his statement, to deaths from ear complications), and it still presents considerable diagnostic difficulties. For example, Mlechin[3] established the fact that out of every 100 cases of death from postanginal sepsis, in only 20 was the complaint diagnosed during life. These diagnostic difficulties can be explained by the fact that with postanginal sepsis there are not always clear signs of it. In diagnosing, it is necessary to rely on the previous medical history of the case, the state of the tonsils, the appearance of general sepsis, and on certain other auxillary signs.

Claus[4] stresses the great diagnostic importance of tenderness of the mandibular angle. The very important factor that in between the commencement of angina and the former appearance of sepsis there may be an interval of some duration, is stressed only by very few (Claus,[4] Hybasek[5]).

Certain difficulties are met with in establishing the side from which the sepsis emanates. It is generally assumed that where the complaint is more clearly indicated, e. g., where there has already been a peritonsillar abscess, there the source of illness must be sought. It is only during the last twenty years or so that postanginal sepsis has been given a closer attention. Abt[6] stresses the importance of the anatomical condition in the spread of postanginal sepsis, and many theories about the origin of the malady as based on this (Hybasek,[5] Claus,[4] Fraenkel,[7] Uffenorde,[8] etc.).

Among the various cases of postanginal sepsis observed by the author,[13] there were two where the illness developed in an unusual

manner, and which are difficult to explain by the theories of the above mentioned authors. In one of them there was sepsis which emanated from the side opposite to that where there had been a peritonsillar abscess. In the other case there was, at the beginning, a typical appearance of postanginal sepsis, but this disappeared, as it were, and together with it the whole inflammatory process as well, and only after a two weeks' period, during which the patient felt quite well was there a sudden reappearance of postanginal sepsis, without, however, any change in the tonsil.

Case 1.—A woman patient, aged 35. She had angina complicated by a peritonsillar abscess on the left side. The abscess was lanced several times and a small quantity of pus evacuated. Temperature was high and irregular. On June 6, 1938, eight days from the beginning, the patient was admitted to the hospital. The clinical history shows that the patient had many shivering attacks with high intermittent fever. Pulse 150 to the minute—weak. Appearance of general heavy septic intoxication. Pain on swallowing. Albuminuria; haematuria.

Examination of the throat showed an open peritonsillar abscess with a large discharge of pus. Right tonsil almost normal in appearance. A slight tenderness of the mandibular angle on both sides when touched. The same day a number of prominent doctors in conference could not decide exactly from which side the sepsis came, and it was decided to wait another day, keeping the patient under observation. By then, however, the tenderness of the right mandibular angle had increased, and a noticeable swelling appeared on this side around the mandibular angle and in the submaxillary region, while on the left side the tenderness had disappeared. On June 8th the operation was performed.

The incision, under local anesthesia, was made along the anterior border of the right sternocleidomastoid, and a ligature placed on the internal jugular and facial veins. The vein was punctured but no blood was found. The upper part of the internal jugular vein, together with the common facial and anterior facial veins, was removed. Examination showed thrombophlebitis in the jugular vein, starting in the anterior facial vein.

Some days after the operation the shivering attacks ceased, and the pulse improved considerably. However, the patient had a metastatic abscess of the lungs and several abscesses of the muscles, and after three months of lying in the hospital she died of general weakness.

On the basis of this case, which is, according to available literature, a very rare one, the following conclusions may be drawn: (1) Thrombophlebitis does not always have its origin in pus collection. The general view that septic thrombophlebitis always comes from the same side as the peritonsillar abscess has its exceptions, and it is necessary, in cases of peritonsillar abscesses complicated by sepsis, to pay attention to the other tonsil as well. E. Simon[9] has described a similar case of sepsis postanginas, where there was a large abscess on the left side. Autopsy findings were: Thrombophlebitis on right side beginning from the facial vein and proceeding to the jugular vein. On the left side, where the abscess was, the walls of the veins were unaffected. (2) In this case, the only way by which it was possible to establish the side from which the septic thrombophlebitis came was from the tenderness and the slight swelling of the mandibular angle. These signs are the chief diagnostic means in such cases. (3) The experience gained in this case shows that the indication to abscess-tonsillectomy recommended by Ganuyt,[10] Link[11] and Heindl[12] must be limited, because the sepsis may not come from the abscess.

CASE 2.—Woman patient, aged 21 years. In March, 1932, she became ill with angina and was brought in on the third, suffering from a peritonsillar abscess. This was lanced and pus found. Temperature was high, oscillating and of a septic character. Pulse weak. There were frequent shivering fits, and there was tenderness and infiltration along the carotid sheath. The jugular vein was palpable. The clinical description showed sepsis caused by the peritonsillar abscess. An operation was proposed but the patient rejected the idea and, contrary to all expectation, the septic condition disappeared and the patient discharged in good health. There were no signs on the tonsils and the pain in the neck had gone. Two weeks after the patient had recovered she had a sudden rise of temperature, shivering fits, and tenderness and infiltration along the carotid sheath passing down to the mediastinum. She died forty hours later showing no signs of general sepsis.

The postmortem showed septic thrombophlebitis starting in the venous plexus of the tonsillar bed and continuing to the cava superior vein, and many septic metastatic lesions over the whole body, lungs, kidneys, etc.

In connection with this case the following peculiarities may be pointed out: (1) It is rare for septic attacks to be interrupted by a period lasting two weeks, during which the patient feels quite well.

In literature dealing with septic angina similar cases are practically non-existent. (2) It is very difficult to explain the reasons for such a rare development of the illness. It may be assumed that the process which had practically passed off, broke out again under the influence of some unknown agent, and that this was situated in the wall of the vein. (3) It is a very good example to show that such cases must be operated on.

The two cases described in this paper go to supplement experience of postanginal sepsis. There are several existing theories about the causes of septic thrombophlebitis. Fraenkel[7] considers that the inflammation comes direct from the small tonsillar veins, while Uffenorde[8] sees the cause in lymphadenitis of the lymphatic vessels and following the course of the facial and jugular veins. Advancing again from these glands, the infection may attack the walls of the veins. Hybasek[5] and Claus[4] both suggest that the sepsis emanates from an inflammatory process (interstitial phlegmon) in the parapharyngeal space. The majority consider that the primary suppuration around the tonsil is the origin of septic thrombophlebitis, and endeavor to give appropriate treatment.

The two cases described here have shown that septic thrombophlebitis may develop independent of, or apart from, the primary suppuration from the tonsils.

Our cases are confirmed clinically by L. Haymann[1, 2] who, on the basis of a large number of autopsy findings on persons who died from septic angina, states: "In a large number of cases the tonsils were inconsiderably affected, and in many, no purulent source from where the infection might have emanated was found." He further established the fact that in 30 per cent of cases the tonsils were not affected at all.

On the basis of the autopsy findings of Haymann and others, one may arrive at the conclusion that the many peculiarities of septic angina are a good reason for further investigation. It may be assumed that the mechanical theory of the cause of septic angina is not sufficiently convincing, and that some unknown biological factors play their part in the appearance of septic angina,[13] giving rise to difficulties in diagnosing and therapy; neither can the author's two cases be explained by the mechanical theory of septic angina. This leads to a search for other causes of septic angina, which, however, for the time being, can only be supposed.

SUMMARY

From the foregoing description of two rare cases of postanginal septic thrombophlebitis we may sum up as follows:

Case 1. The 35-year-old patient became ill with a peritonsillar abscess after she had had angina, but septic thrombophlebitis developed on the opposite side where the tonsil showed no signs of being affected.

Case 2. The 21-year-old woman became ill with septic angina, after which there was a two week interval during which she felt quite well. She then had a fatal relapse.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 6, 1939

THE PRESIDENT, DR. GEORGE T. JORDAN, IN THE CHAIR.

Surgical Repair of Facial Paralysis

HANS BRUNNER, M.D.

CHICAGO

(Author's Abstract)

The anastomosis of the facial nerve and the nerve grafting will be exhaustibly discussed and the disadvantages of both methods pointed out. The author recommends two methods which avoid the disadvantages of the nerve operation to a great extent. His procedure is as follows: First, he does the endoral muscle plug operation. The masseterian muscle is exposed within the mouth and is pulled to the angle of the mouth through a tunnel of mucous membrane. When the wounds in the mouth are healed, the plastic will be completed by a subcutaneous lifting of the angle of the mouth. The following procedure is employed: incision in the mouth as in a Denker operation, exposure of the canine fossae; with a drill a hole will be performed in the piriform aperture and a silk thread will be put through the hole. Both ends of the silk thread will be sewed on the angle of the mouth and on the philtrum, respectively, and both ends will be tied between the angle of the mouth and the philtrum towards the piriform aperture and to approximate the angle of the mouth to the philtrum.

DISCUSSION

DR. PAUL W. GREELY: I was pleased to receive this invitation to discuss Dr. Brunner's paper, because it gave me an incentive to hear him. I must confess his type of operation is new to me. I can

see that it offers many points. First, however, I would like to take up some of the older types of operation, which I think still have merit.

I really feel that all cases of facial paralysis should be considered from the standpoint of the nerosurgeon. As he pointed out, these operations are not overwhelmingly successful. If one can anastomose the ends of the facial nerve that method should be given consideration. It has been the experience of practically everyone who has done nerve transplants that the results are far from satisfactory. If neurosurgery cannot be utilized one then has to resort to some other method.

Probably one of the older methods that has merit is transplants of fascia lata. This does not require a large incision. I do not believe that the production of scars on the side of the cheek is any great matter. The incision can be above the hair line and be quite inconspicuous, and with a needle, strips of fascia lata can be brought down and transplanted. You must use enough strips to lift up the side of the face adequately. One important point is to bring one strip to the angle of the mouth, to lift the side of the mouth, and one should be placed at the angle of the nose. One should then be placed at the philtrum and another in the lower lip. If they are passed just beyond the midline, the functional result is better than if proximal to the midline.

Another point which is very useful, which Ferris Smith has pointed out to me is to place strips around the other angle of the mouth to give more lift to the lower lip, thus eliminating the tendency toward drooping. If the strips of fascia lata have been properly placed and sutured, they can be pulled up through the small temporal incision and brought beyond the point of over-correction, and the ends then sutured to the parotid fascia. The patients are usually much more comfortable even though it leaves the side of the face expressionless and masklike.

Another procedure Dr. Brunner pointed out is to use part of the temporal muscle. If one uses a large flap of temporal muscle, he is going to get a bulge, and it is necessary to use a large flap to get enough strength, but I think if a smaller piece is used a lot of that bulge can be avoided. In fact, one can make a groove in the zygomatic arch to hide it. Furthermore, small strips can be brought up to the upper and lower eyelid to get closure of the lids. I think these are an advantage, but they are not strong. Although you may

see photographs where the patient can smile, they tend to tire, and the expression changes.

I think, therefore, that this type of operation, plus strips of fascia lata, has much to offer. After the fascia lata strips are placed, a small temporal flap of temporal muscle is placed to produce activation.

Dr. Brunner's operation appeals to me because of its simplicity. I have but one criticism to make. I have always been skeptical about plastic procedures where one goes through the mouth. I am afraid of carrying infection into the cheek, with subsequent loss of the effect one desires. I am interested in Dr. Brunner's contribution and want to see more of it, but I am a little fearful of infection. When we pass strips of fascia lata subcutaneously we are very careful that they do not penetrate the buccal mucosa for fear of infection. I would like to have him answer this point.

DR. THOMAS C. GALLOWAY: In these cases of facial paralysis the greatest worry is not the asymmetry of the features, but what happens to the uncovered cornea, because it is so easy to get a red lacrimating eye and even a corneal ulcer and loss of sight. For this reason simple facial plastics cannot give the result that restoration of movement and tone through nerve grafting does. Such a demonstration as Sullivan gave at Toronto with numerous successful cases makes it seem that simple facial plastics hardly can offer the same possibilities. And knowing this, there should not again be the number of old untreated paralyses that will respond only to plastic procedures, but rather these cases should have nerve grafting before fibrosis and atrophy are established. However, as Frazier established, one should probably wait at least six months for spontaneous recovery before resecting nerve scar. We had one bullet injury, with comminution of the mastoid followed by suppuration, in which function began to return only after eight months.

This demonstration by Dr. Brunner is ingenuous and carefully worked out and surely will be valuable in proper old cases.

DR. ALFRED LEWY: There is another operation which was not mentioned, that devised by Bunnell of San Francisco. He is a general surgeon. He does an end-to-end anastomosis of the seventh nerve, uncovering beyond the stylomastoid foramen if necessary, to take advantage of the curvature forward that takes place at that point. He gets enough space to make a direct suture without nerve

graft. The operation is worthy of mention. I do not know the results except from the author's claims.

Dr. Joseph C. Beck: I do not believe there are many in this audience who recall the earliest surgical work done on facial paralysis or repair by the late John B. Murphy. It was his contribution to surgery of the face in using the spinal accessory in the repair, with a successful result so far as appearance and motion were concerned. But unfortunately, as he stated, when the patient carried an umbrella and moved the arm the face moved at the same time. That brought the operation into disrepute and it was not long before the spinal accessory was given up. Then came the repair work with the hypoglossal nerve, and during these years I did only one spinal accessory operation and the patient had the same movement of the shoulder associated with the face. I did several operations with the hypoglossal anastomoses, both end-to-end and end-to-side. I would get as much as possible of the facial nerve out of the bony canal and attach it to the side of the hypoglossal without cutting the nerve to the tongue. Consequently one did not have this objectionable palsy of the tongue.

The main feature in nerve plastic is to learn how to unite the nerve. Dr. Fraser, the Englishman who first brought out the operation that Dr. Lewy called attention to tonight, showed that the secret of the success of this work was the way in which the perineurium was sutured. I had the great satisfaction of having been taught to suture nerves by the great Alexis Carrel, when he first came to this country, and to have been taught by a man who knows as well as he does is quite a treat. Now as to the affection about the cornea, mentioned by Dr. Galloway. I also want to bring that point out, and say that a simple procedure advanced by the late Dr. Arthur Prince of Springfield, Illinois, has been and still is practiced and has saved many an eye. A small adhesion was allowed to form between the upper and lower lid at the outer one-third side of the eye, not enough to obstruct vision, but sufficient to bring up the lower lid to protect that cornea.

I have had no experience with the operations recommended by Ballance and Duel, but I think Dr. Kulvin and Dr. Russell, both of whom are present and have done this operation at Hines Hospital on two cases, will not hesitate to give us their results.

I also want to say that I think muscle plasty shown by Dr. Brunner should be a great encouragement in this repair work. He

made one point in particular, that is, that we do not know how much recovery we will still obtain if we do not cut the nerve. One well-known French surgeon has shown that eighteen years after a paralysis of the facial nerve had existed spontaneous recovery may take place. The operation shown here by Dr. Brunner for lifting the angle of the mouth, I think is a wonderful contribution. I do think, however, that when we go through an infected area as he advises, is a mistake, and why should we do this in this operation when we can make a small incision through a clean field externally?

Dr. Roland Russell: Unfortunately the patient upon whom I operated passed from my observation and I never did know the result of my one and only case. You spoke of causing deafness by this operation. A large number of these patients are already deaf, because the paralysis occurred from a radical mastoid operation. As to the great scarring produced by this operation, most of them already have a mastoid scar, and even if they did not, the scar is mostly behind the ear and is not conspicuous.

Dr. Max Kulvin: I did a Ballance-Duel operation for facial paralysis. Improvement began in three to four months. The patient noted the difference in his features. Moving pictures taken one year later revealed gradual improvement.

Dr. F. B. Moorehead: I like the clever way in which Dr. Brunner handled the matter of nerve anastomosis, and I agree with him thoroughly. There may be successful cases, but I have never seen one which involved the seventh nerve. I would like to elaborate a bit more on what Dr. Greeley discussed, that is, fascia lata suspension. Unless it is Dr. Brunner's new operation, I believe the fascia suspension offers the best result. The failure of the fascia lata operation lies in the angle at which the suspension is placed. To suspend the angle of the mouth, the fascia suture should be in a horizontal line. When it is placed back at the angle Dr. Greeley indicated, it will not be as successful. Usually three suspensions, one placed at the angle, one midway between the angle and the wing of the nose, and the other one close to the wing of the nose, will be sufficient.

If there is an equal showing of the vermilion border, then the sutures are best placed in the nasolabial fold. If, on the other hand, there is insufficient showing of the mucous membrane, a suture should be brought close to the muco-cutaneous border. The entire

lip may be suspended with three long suspensions and the border everted with two or three short suspensions. As Dr. Brunner pointed out, the suspension of the lip to a point of symmetry is of benefit only when the face is in repose.

DR. FRANCIS LEDERER: The thought occurred to me, would it be possible to use fascia lata for this triangular suture instead of the other type? Then, with reference to infection, it has been my experience so far, that the best results have obtained where there has been a low-grade infection. I have seen good results in lid ptosis with low-grade infection. It aids traction. I do not see any great objectionable feature in working through these areas and taking a chance of having some low-grade infection present.

DR. HANS BRUNNER (closing): First, I wish to thank you for your interest. I wish particularly to thank Dr. Moorehead and Dr. Greeley for this instructive discussion, since the subject of my paper touches the borderline of otolaryngology in only certain details. I want to say that the plastic mentioned by Dr. Greeley is one which I did three times, once using silver wire and twice using fascia lata. In all three cases phelgmons occurred and the fascia lata as well as the silver wire slipped out. This experience taught me the danger of working in the surroundings of the mouth subcutaneously, and that is also the reason why I have given up this kind of practice.

To Dr. Galloway's question, I wish to say that it is, of course, very important to protect the cornea. That can be done either by a tarsorrhaphy or by a muscle plastic from the temporal muscle. I did not mention this procedure because I could not add anything new to that method. I know of the paper of Dr. Sullivan, and so far as I remember, it does not contain any pictures of patients, and I believe that in the cases of Sullivan the tic-like movements appeared after the nerve grafting.

With reference to Dr. Lewy's comment, I want to say that the pictures I demonstrated from the case on whom the expressional movements of the face were returned, is the case of Bunnel. I think you agree that the results in that case cannot be considered ideal.

I thank Dr. Beck for his historical remarks, and I think that as far as the protection of the cornea is concerned, I have answered his remark. The technic of suturing a nerve is very delicate. The suture goes through the sheath of the nerve only.

To Dr. Russell and Dr. Kulvin, I have to repeat that in cases on whom a facial palsy occurs after a radical mastoid operation, the question as to what to do is much more easily answered. However, I had in mind particularly those cases on whom the facial palsy appeared, for instance, after simple mastoid operation or on a rheumatic basis, etc. I wish to repeat that in those cases the procedure of Ballance and Duell present the danger of impairment of hearing.

To Dr. Fishman, I would say that neurotization means that the nerves grow from the transplanted masseterian muscle into the paralyzed facial muscles. In that way the ingrowth of the regenerated facial nerve fibres into the facial muscle is facilitated.

I agree with Dr. Moorehead about the installation of the mechanical suspension. When the traction on the end of the mouth is directed toward the posterior end of the zygoma, as is usual, we may elevate the angle of the mouth, but simultaneously we also enlarge the width of the paralyzed half of the upper lip. This we do not want since with a facial palsy the width of the paralyzed half of the upper lip is enlarged anyway. It is, therefore, much better to direct the traction on the lower lip perpendicularly upward, by which traction the end of the mouth can be intensely elevated without enlarging the width of the upper lip. This is just what I am trying to do with the subcutaneous lifting of the angle of the mouth, which is directed, as you have seen, towards the piriform aperture, viz., to the midline.

Finally, I wish to say to Dr. Lederer that in the subcutaneous lifting of the mouth, I do not use fascia lata, since with my experience fascia is very sensitive against infections and even against mild infections. I do, however, absolutely agree with Dr. Lederer concerning what he said about mild infections in plastic surgery. This is particularly true in these plastic procedures I have discussed tonight. Although that statement seems to be somewhat paradoxical it can be very easily explained, since these mild infections which accompany the endoral approach yield subcutaneous scars which are, of course, helpful in the correction of the deformity, and this is one of the reasons I prefer the endoral approach. The other reasons are that this procedure, as I have already said, avoids the scars in the skin, and furthermore, infections which occur after subcutaneous operations, viz., external operations, are in plastic surgery much more dangerous than infections occurring after submucous operations, viz., endoral approach. This answers the objections of Drs. Greeley and Beck.

Giant Follicular Hypertrophy of Nasopharynx: Report of a Case

FRANK J. NOVAK, JR., M. D.

(This paper is printed in full on page 526 of this issue.)

DISCUSSION

DR. JOSEPH C. BECK: This patient was referred to me by another physician, who telephoned and said he was sending a fellow down and "to blow him up," meaning inflation. He said that was all he needed and it would not be necessary for me to examine him. I proceeded to do that, but after getting the history, examining the man, and looking at the nasopharynx, I felt there was something unusual. I thought it might be the remains of an adenoid. When I found he had been very thoroughly examined elsewhere and a piece of tissue removed, I telegraphed for the findings. Very promptly I received a complete history of the case, except that the report on the tissue removed was not complete. All it said was that it was not malignant. I did try to inflate the middle ear, passed a catheter, but it was impossible. There was no change in the tympanic membrane: the hearing was definitely indicative of a middle ear deafness. I then had the patient completely examined by the same physician who sent him to me. I told him since that great clinic did not find the growth it might be that we were dealing with a disease in which blood changes might be found. The report came back that the blood examination was negative and the physical examination normal, except that the man was very fearful because we had made such a to-do about his case. I passed a rubber uretheral catheter through the nose and drew the palate forward so as to get a better exposure. The first biopsy made was not satisfactory. The pathologist would not commit himself and was in doubt. Dr. Novak and I decided that the growth should be entirely removed and further studied. You have heard the clinical side from Dr. Novak, who performed the operation. I want to emphasize the point that the x-ray treatment given preoperatively for diagnosis had no effect on the growth whatsoever. Dr. Novak and I studied the preparation together and peddled the specimen around to other pathologists, because it was so unusual. As you heard from Dr. Novak, it is a rare lesion. I wish you could have looked into the microscope to see what could not be shown on the screen.

I have here a picture postcard of this man, taken in Mexico about three weeks after the operation. He got immediate relief from the total occlusion, there was perfect ease of inflation, and so far he

does not speak of any recurrence. At no time were there any glands in the neck; it surprised me, because with a mass like that, if he had lymphoepithelioma or lymphosarcoma or a blood dyscrasia, he should have glands in the neck.

Polypoid Granuloma of the Larynx Following Endotracheal Anesthesia

WILLIAM A. SMILEY, M.D.

(Abstract)

In reviewing the literature the essayist has been able to find but three cases of polypoid laryngeal disease following endotracheal anesthesia.

The case herein reported was that of a woman, aged 35, upon whom operation for stenosis of the common bile duct had been performed under endotracheal cyclopropane. On the tenth postoperative day she complained of hoarseness, and laryngeal examination showed a small polyp in the left side of the larynx. Removal was advised, but was refused until about two months later. Microscopic diagnosis was of granulation tissue with acute and chronic inflammatory changes (pyogenic granuloma). The polyp was a soft rounded mass about 1.5 cm. from above downward, about 1 cm. in transverse diameter, and was pedunculated.

It seems justifiable to conclude that this subglottic granuloma was a result of trauma from the endotracheal anesthesia. It is suggested that the larynx may react to trauma by the formation of an infectious granuloma such as infrequently found on the nasal septum.

DISCUSSION

DR. THOMAS C. GALLOWAY: I wish Dr. Smiley would tell us how polypi are produced in the trachea. I have recently had a very trying case in which one lung had been collapsed for tuberculosis, and the trachea and remaining bronchus were so filled with polypoid masses that the patient went into asphyxial convulsions before I could get a bronchoscope through to reopen the lumen. After removing these in three sessions with cup and biting forceps, they proved to originate from an ulcer about 2 cm. above the carina. They were not covered by epithelium, but were masses of simple edematous granulation tissue. Apparently their weight and the traction of

coughing and gravity had pulled them some distance from their point of origin. Probably as they elongated they filled with edema fluid and their weight then gave more pull by gravity. With the base of the ulcer superficially coagulated their recurrence seems stopped.

DR. B. B. LENNON: This patient, classed as a No. 3 risk (distinctly not a good risk), was scheduled to have a choledochoduodenostomy. Her blood pressure was 130/74, pulse 120 before anesthesia. Cyclopropane was administered at 300 cc. per minute, with adequate oxygen. Ten minutes later, the patient was intubated; intubation was uneventful. The patient was maintained in first and second plane of surgical anesthesia for an hour and twenty-two minutes, during which time the blood pressure varied from 140/94 to 120/82, pulse from 130 to 84. The patient was awake a few minutes after anesthesia and operation, and retched upon recovery. She was returned to bed in fairly good condition and was seen the same day in the afternoon.

I note on her postoperative follow-up that she experienced nausea-emesis on the operative day and the first day postoperative, none thereafter. However, she complained of hoarseness upon the operative day and every day thereafter for some time. As the days passed and the hoarseness persisted and even became worse, a question arose in my mind as to cause. I had heard of the case of Cohen as reported by Dr. Smiley, some months previously and the question of a polyp began to raise itself in my mind. An ear, nose and throat consultation was obtained and laryngoscopic examination was done thirteen days postoperatively, with positive findings.

Intubation was effected with a Guedel laryngoscope and a latex wire woven catheter, using a celluloid knitting needle as a stilette. The stilette in its entirety was covered with a thin layer of sterile petrolatum as was also the end of the endotracheal tube and the Waters-Guedel inflatable cuff.

Adequate lubrication facilitates the introduction of the tube between the vocal cords with a minimum of friction. The three cases that have been reported in the literature all followed nasal intubation with a soft catheter of the Magill type. This case is the first one to follow introduction of a latex rubber catheter. This catheter has a spiral wire of stainless steel in its wall to maintain its shape and lumen. When this patient's larynx was exposed, the airway was free and intubation was effected with ease. No growth or abnormality was noted in the pharynx, larynx, vocal cords or prox-

imal portion of the trachea. This procedure takes from 10 to 30 seconds on the average, and in this case was probably the former, because it was effected with no technical difficulty. I am quite sure there was no polyp present before operation.

Like many things in medicine, we physicians sometimes never realize the value of certain procedures until they have lapsed into disuse and have been resurrected one or more times. An excellent example of this is the development of the use of ether. Although it was used in so-called ether frolics and at parties for stimulation, it was not used for anesthesia until 1840 by C. W. Long. It had, however, been used in the treatment of asthma at the Davey Pneumatic Institute in England in 1785. Historically, laryngeal intubation is older than general anesthesia. Fine of Geneva practiced it in 1800 and Dessault in 1801. In 1867 laryngeal intubation during anesthesia was practiced by Trendelenberg. It was not until the last generation that endotracheal anesthesia really received its impetus, as a result of demands put upon anesthesia by developments in various branches of surgery. This technic of anesthesia had aided considerably in the development of chest surgery and unquestionably facilitated operations around the head and neck.

It allows perfect ventilation and prevents laryngeal spasm. There is no tax on the respiratory mechanism of the patient, respirations being free and under control. Patients are protected against the entry of blood, mucus and purulent secretions into the trachea. Aspiration of tracheal and bronchial secretions is performed easily. It facilitates resuscitative procedures and the use of artificial respiration.

Its disadvantages are dependent to a great extent upon the skill of the anesthetist. Since instrumentation is required, there is danger of trauma to the teeth, lips, soft palate, tonsils, epiglottis and vocal cords.

This procedure has been performed many thousands of times by hundreds of individuals with little distress to the patient. At Michael Reese this technic has been utilized in over 100 cases of all types, and has been carried out by members of the anesthesia staff several hundreds of times at other hospitals in the past years. It has been used at Michael Reese for tonsillectomies in adults twelve times. With such a technic, a great burden is lifted from the surgeon's shoulders and the sense of security in these cases cannot be minimized.

In comparing the value of a wire woven catheter with a Water inflatable cuff and a Magill soft rubber tube, one can readily see that the cuff can be blown up like a balloon, thereby closing off any possible means of material invading the respiratory tract from the gastrointestinal tract, or of contaminants in the upper respiratory tract finding their way into the lower respiratory tract. This might well have happened in a number of cases, not only during abdominal operations but also in operations around the head and neck, were it not for the presence of the inflated cuff. Normally there is present 0.1 normal hydrochloric acid in the stomach and we know that experimentally the injection of small amounts of 0.1 normal hydrochloric acid into the trachea and bronchial respiratory tract of a cat will produce lung abscess, necrosis and pneumonia. It can be readily seen that in certain types of abdominal operations where manipulations of the stomach has caused patients to vomit large quantities of material which could not find their way into the lower respiratory tract because of the presence of this tube and cuff, that it has probably served to save the life of many a patient. The Magill tube depends for its efficiency to some extent upon the tonicity of the cords and the constrictor muscles of the trachea. It is conceivable that material that has been vomited may be able to run down between the tube and the trachea, thus finding its way into the lower respiratory tract.

Dr. William A. Smiley (closing): I want to thank Dr. Lennon and Dr. Galloway for their discussion.

With reference to Dr. Galloway's case, this patient did not present a true polyp, in that it did not spring from glandular elements. This is granulation tissue as shown by development of blood vessels, the enormous amount of leucocytes, with more or less normal epithelium lying over the polypoid granulation. The case Dr. Galloway mentioned apparently has one common feature with this, that is, in his case the granulation tissue arises from a small ulcerated area. Probably this polypoid granulation also arose from a small ulcerated or traumatized area. The patient showed no allergic manifestations.

Notices

Tenth Annual Meeting of the Biological Photographic Association

The tenth annual convention of the Biological Photographic Association will be held at the Hotel Schroeder, Milwaukee, Wisconsin, September 12, 13 and 14. This society is interested in the study of photography as applied to the biological sciences, and the improvement of its technic. Scientific photographers from all parts of the country will meet to exchange ideas and information on still and motion picture photography including the latest developments in color work. Formal papers will be augmented by round-table discussions. A salon of natural color and monochrome prints of biologic and clinical subjects illustrative of the highest type of biologic photography will be a feature of this meeting.

The membership of the Biological Photographic Association is recruited from professional, clinical and biologic photographers as well as physicians, dentists and other scientists interested in this specialized branch of photography.

Communications may be addressed to Anne Shiras, Secretary, University Office, Magee Hospital, Pittsburgh, Pa.

Eighteenth Annual Summer Graduate Course in Ophthalmology and Otolaryngology

Denver, Colorado

July 29th to August 10th, 1940

This Pioneer Summer Course presents clinical lectures and demonstrations by men of national prominence in each specialty. The first week is devoted to Ophthalmology and the second to Otolaryngology. Sponsored by the Colorado Ophthalmological and the Colorado Otolaryngological Societies. Fee, $50.00, for both weeks.

Registrar: Dr. H. L. Whitaker, 1234 Republic Building, Denver, Colorado.

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